







Emergencies: saving more lives in your practice



Amanda Boag René Dörfelt Isabelle Goy-Thollot Chiara Valtolina



Emergencies: saving more lives in your practice



Table of contents

	The authors	3
	Introduction	5
1	Initial trauma case	6
2	How to make your practice "emergency ready"	9
3	Shock and fluid therapy	16
4	Approach to the dyspnoeic patient	24
5	The vomiting emergency patient	34
6	Do's and don'ts	43
	Further reading	54



The authors



From left to right: Chiara Valtolina, Isabelle Goy-Thollot, René Dörfelt and Amanda Boag.

Amanda Boag

Amanda graduated as a veterinary surgeon from Cambridge University in 1998. She undertook further clinical training at the Royal Veterinary College and the University of Pennsylvania, and is Board certified in both Internal Medicine and Emergency and Critical Care. She was a Lecturer in Emergency and Critical Care at the RVC from 2003-2008. In September 2008, she took up the post of Clinical Director at Vets Now where she has responsibility for clinical and professional standards and training across 53 emergency clinics and two 24-hour hospitals.

She is the author of many peer-reviewed articles and book chapters and is co-editor of the BSAVA Manual of Emergency and Critical Care. She acts as a veterinary consultant for Pet Blood Bank and has an active interest in transfusion medicine. She is founding president of the European College of Veterinary Emergency and Critical Care (ECVECC),

and past president of the European Society of Veterinary Emergency and Critical Care (EVECCS). She is an elected RCVS Council member since 2012 and is currently Treasurer of the RCVS. She received the BSAVA Melton award for meritorious contribution to small animal practice in 2011.

René Dörfelt

After graduating from the Faculty of Veterinary Medicine at the University of Leipzig, Germany, René undertook an Internship and wrote his dissertation thesis on haemodialysis at the Small Animal Clinic of the Free University of Berlin. While working at a large private animal referral hospital in Hamburg, he was responsible for the management of emergency, critical care and dialysis patients. From 2007 to 2011, René undertook residency training in Veterinary Anaesthesia and Analgesia



at the University of Veterinary Medicine in Vienna, Austria. Since 2011 he has been Head of the Emergency and Critical Care Service at the Clinic for Small Animal Medicine at the Ludwig-Maximilians-University-Munich. In 2012, he became a Diplomate of the European College of Veterinary Anaesthesia and Analgesia. He is involved in the European Veterinary Emergency and Critical Care Society, and has authored and co-authored numerous journal articles. He has given over 400 continuing education lectures in the field of Veterinary Emergency and Critical Care as well as Anaesthesia and Analgesia.

Isabelle Goy-Thollot

Isabelle Goy-Thollot is responsible for the ICU at the Vet School of Lyon, France (SIAMU). She graduated from Maisons-Alfort, France, in 1989 and completed a PhD in 2005 on the hypothalamo-pituitary-adrenal axis. She was a Lecturer in Internal Medicine before founding the SIAMU (SA-ICU) in 2002. She is EVECCS past president and currently chair of the EVECCS scientific committee. She has been an ECVECC Diplomate and invited specialist since June 2014. She is Chair of the ECVECC credential committee. She is interested in nephrology, dialysis and blood transfusion. In her spare time, she likes dancing, reading thrillers and going trekking.

Chiara Valtolina

Chiara graduated in 2000 from the Faculty of Veterinary Medicine at the University of Milan, Italy. She worked for 4 years at the Small Animal Surgery Department of the same faculty undertaking a research doctorate. In December 2004, at the end of her doctorate, Chiara began an externship programme at the Intensive Care Unit (IZA) of the Department of Clinical Sciences of Companion Animals at the Faculty of Veterinary Medicine, Utrecht University, in the Netherlands. In June 2006, Chiara started her residency in Emergency and Critical Care at the Royal Veterinary College in London. She completed her residency in June 2009, and, in September 2009, she became a Diplomate of the American College of Emergency and Critical Care.

Since November 2009 she has worked as a staff clinician and Lecturer in the Intensive Care Unit (IZA) of the Department of Clinical Sciences of Companion Animals at the faculty of Veterinary Medicine in Utrecht. Cats have always had a special place in her professional life. During her residency in Emergency and Critical Care, Chiara focused on managing traumatized animals.



Introduction



Every practitioner can be faced with emergencies, and the decision you make in the first few hours can be the difference between life and death for your patients. Although some practices focus on emergency care and specialist emergency and critical care units exist, the majority of emergency patients are seen by veterinary surgeons just like you... and you can make a huge difference.

Treating emergencies well is important not only for the patient but also for you and your clinic team. Saving a life and knowing you have done a good job is a great morale boost for your staff, and impresses itself indelibly on the owner's mind. In terms of reputation for you and your practice, the benefits of managing an emergency well can be high.

The best way to reduce the stress linked to emergency situations is to be well prepared. Preparation involves both having the right equipment close to hand and having thought through what you might need to do. Other professionals that work with emergencies such as firemen and policemen do real-scale exercises in order to identify potential inefficiencies and find ways to

prevent them. This habit could be a great idea in veterinary medicine.

This *Veterinary Focus* Special Edition has been made by four specialists in Emergency and Critical Care. They have all benefitted from long periods of training and reflection on their own mistakes as well as working with many colleagues in general practice and seeing many of the things that can and do go working when faced with making urgent decisions in stressful situations. Our choice has been to focus on the most frequent emergencies and, on the main things, we wish we knew when we started out. We hope it will help you to get better prepared.



Philippe Marniquet, DVM, Dipl. ESSEC Royal Canin



1. Initial trauma case

> SUMMARY

Let's start with a real case that could come through your practice doors today. Working through a real case will highlight all the important practical and emotional issues we need to consider when dealing with real patients.

You are about to begin your night shift when you receive a telephone call from a very concerned owner: his dog Lucy, a three-year-old neutered female Jack Russell Terrier, has been the victim of a road accident. The owner who was at the scene reports that, after the impact, Lucy tried to get up, took a few steps and then collapsed. She is now breathing badly and seems to be in a lot of pain. The owner is already on his way and should reach the clinic in about 20 minutes.

1/ Are you ready to deal with an emergency trauma case?

Read on and we will touch base at the end with answers to the questions posed in this first case.

- Are you ready for this emergency?
- What alterations to the cardiovascular, respiratory and neurological systems can you expect to find as a result of the vehicle accident?
- Ask your assistant to begin preparing the rooms for Lucy's arrival.
- What would you need to initially manage this emergency?

Lucy is carried in by her owner, who is obviously in shock and very anxious. You ask the owner to kindly wait in the reception area, and tell your assistant to bring him a glass of water or something warm to drink, while you conduct an initial assessment of Lucy's clinical condition.

The physical examination finds:

- Heart rate of 160 bpm
- Weak pulse

- Pale mucous membranes
- Capillary refill time > 2 s
- Respiratory rate of 40 bpm
- Dull pulmonary sounds, dorsally and bilaterally
- Depressed mentation status
- Palpation of abdomen: painful
- Body temperature: 36°C
- Visible haematoma and crackling noise on palpation of left femoral region (Figure 1)
- What conclusions do you draw with this initial assessment? Is Lucy in shock? What type of shock? How do you recognise this condition?
- What is the best initial therapy approach to stabilise Lucy? If you decide to stabilise her using fluid therapy, which fluid would you choose? How much fluid would you administer? At what rate?
- What is the most probable cause for dyspnoea in Lucy?
- Do you think that administering oxygen to stabilise her will be enough?
- Your assistant suggests administering an analgesic because Lucy is in a lot of pain. What do you think about using a non-steroidal anti-inflammatory drug (NSAIDs)? Or perhaps you would prefer an analgesic from the opioid family?

The owner is impatient, in shock and still waiting in the reception area. How do you manage the owner? What information is it important to provide to the owner before diagnosing and treating Lucy?

Lucy is obviously just one example of the types of emergency that could arise during your shift. On reading this case, do you feel prepared to offer Lucy the best possible stabilisation, considering her clinical condition?







Figure 1. Lucy on admission: note the lateral recumbency, cardiovascular collapse (a) and the visible haematoma in the left femoral region (b).

2/ Answers to the first case

Patients like Lucy, suffering from trauma as a consequence of a vehicle accident, are considered polytraumatised, because they often suffer acute alterations in relation to several body systems, especially the cardiovascular, respiratory, neurological and skeletal ones.

Alterations that we can expect as a consequence of blunt trauma include: cardiovascular shock and tissue haemorrhage; dyspnoea secondary to pulmonary contusions, pneumothorax and/or haemothorax; neurological alterations as a result of spinal fractures and cranial trauma; haemoabdomen; skeletal fractures and injuries to the soft tissues. If we are aware of the imminent arrival of a patient that has been the victim of a road accident, we need to ensure that our rooms are equipped as best as possible to manage the emergency (see **Chapter 2**).

We will need:

- Clippers
- Alcohol and chlorhexidine to aseptically prepare the skin
- Varying sizes of intravenous catheters
- Heated fluids
- Infusion sets, infusion pumps or pressure bags (in the case of a medium-large size animal)
- Supplementary oxygen and a circuit for flow-by administration
- Mu-pure category opioid analgesics (methadone, fentanyl, morphine)
- · A kit for carrying out a thoracocentesis
- Material for bandaging
- A monitoring system for an electrocardiogram and assessing the pulse oximetry
- Equipment to perform an emergency database

Patients like Lucy are usually in a state of shock, referred to as traumatic shock, characterised mainly by secondary hypovolaemia with external or internal haemorrhage. Acute haemorrhage can also be associated with fractures to the longer bones and/or acute trauma to the soft tissues as with Lucy's case. In traumatized patients, we need to be prepared to deal with other types of shock, in addition to hypovolaemic shock. The pain associated with trauma can inhibit the vasomotor centre, thus interfering with the peripheral compensatory vasoconstriction response. Acute lesions and trauma to the spinal cord and skull can cause neurogenic shock, with loss of peripheral sympathetic tone. Cardiogenic shock as a consequence of myocardial contusion and arrhythmia could worsen perfusion.

Recognising whether an animal is in shock is performed by conducting a thorough examination of the cardiovascular system, focusing your attention on assessing the perfusion parameters: heart rate, quality of peripheral pulse, colour of the oral mucous membranes, capillary refill time and mentation (see **Chapter 3**). In dogs, the alterations in these parameters vary according to how serious the hypoperfusion is.

Lucy is tachycardic, with weak pulses and pale oral mucous membranes (Figure 2) associated with an increased capillary refill time, as a result of peripheral compensatory vasoconstriction. Lucy is in a moderate state of shock.

The patient that has been a victim of a vehicle accident must be examined quickly, and any alteration to the cardiovascular and respiratory systems must be stabilised. It is important to begin administering flow-by oxygen during the initial physical examination.







Figure 2. Lucy on admission: note the severe mental depression (a) and the pale mucous membrane (b).

An intravenous catheter should always be placed in any unstable animal, so as to quickly administer any treatment such as fluids and analgesia, and to have venous access in case of cardiopulmonary arrest.

The treatment for hypovolaemia requires replenishing the deficit in the circulating volume using intravenous fluid therapy. There are different types of fluids available that can be used for these patients (isotonic crystalloid fluids, colloid solutions, hypertonic crystalloid solutions and transfusions of blood or its products) (see **Chapter 3** "Shock and fluid therapy").

Isotonic crystalloid fluids are often considered as the first choice. These fluids are administered in a bolus manner, and the dose varies according to how serious the hypoperfusion is. In traumatic shock associated with haemorrhage, or when there are pulmonary contusions, administering low volumes 10-20 mL/kg as an initial bolus is preferred with infusions over a short period of time (usually 15-20 minutes).

At the end of the bolus, the animal must be reassessed to see whether there is an improvement in the cardiovascular parameters and whether an additional bolus needs to be administered.

We administer oxygen to Lucy using the flow-by technique and place a peripheral catheter in the cephalic vein. We decide to administer an initial 20 mL/kg bolus of lactated ringer for Lucy. Because the perfusion parameters have not normalised, we decide to repeat a bolus of 20 mL/kg twice in 15-30 minutes; at the end of the third bolus Lucy is stable from a cardiovascular perspective.

Lucy's dyspnoea is definitely associated with pulmonary contusions, but the marked reduction or absence of pulmonary breathing sounds dorsally are due to a pneumothorax. Administering oxygen will not on its own be sufficient to stabilise the dyspnoea in Lucy, which requires thoracocentesis, probably bilaterally (see **Chapter 4** "Approach to the dyspnoeic patient").

Polytraumatised patients suffer moderate to severe pain, and the pain must always be treated during the initial stabilisation stages. The use of NSAIDs is contraindicated for animals in shock or that are not stable from a cardiovascular perspective. We recommend using a pure $\mu\text{-opioid}$ such as morphine or methadone (0.1-0.2 mg/kg IV, IM) for these patients. Opioids are the ideal drugs for treating moderate to severe pain, because they have minimal influence on the cardiovascular system of these patients.

Lucy is suffering severe pain; during the initial stabilisation phases, we decide to administer 0.2 mg/kg of methadone intravenously.

Once Lucy's general clinical condition has been established, it is important to provide the owner with accurate information. Information such as a cost estimate, the proposed diagnostic and treatment plan, and the prognosis must always be clearly discussed with the owner. Consent for possible cardiopulmonary resuscitation must be discussed with the owner.



2. How to make your practice "emergency ready"

> SUMMARY

The most important factor in being able to treat emergency patients successfully is the preparedness of the clinical team. This chapter also reviews the equipment required for an emergency room and emphasises the importance of communication within the team and with the pet owner.

Introduction

Emergency and critical care medicine has become established as one of the newest and most dynamic specialities in small animal practice over the last 20 years. Our capacity to deliver life-saving care to our patients has increased, and our clients expect high-quality care.

Advanced critical care with techniques such as mechanical ventilation and haemodialysis will always be limited to a small number of institutions; however, all practices regardless of their size see patients presenting as an emergency. The definition of a veterinary emergency is usually taken to mean any patient where the owner or carer of the animal is concerned that the animal is acutely unwell. Practically, this means that veterinary emergencies range from animals with very minor problems through to patients that are close to death. Importantly as owners are not trained professionals, the stability of a patient and whether it is a stable or unstable emergency cannot be determined until a trained veterinary professional has examined them.

The ability to deal with emergencies efficiently with the best chance of a good outcome for the patient and owners relies predominantly on the people. Having a well-trained motivated team with good communication both within the team and with the client is the single most important factor. Beyond that, ensuring the physical environment is appropriate with a central well-equipped location within the hospital/practice to triage and stabilise patients, and supports the clinical team in delivering this care. Being prepared is vitally important to achieving a successful outcome for the critically ill. All practices should be able to see an emergency, provide urgent stabilisation and recognise when onward referral to an ICU is desirable. In this chapter, we will explore how to make your practice "emergency ready", ensuring you can provide great care for your patients and their owners.

1/ The team

Veterinary surgeons have prime responsibility for diagnosing and treating animals and act as leaders of the veterinary team. In smaller practices the veterinary surgeon may be working alone, and although it is possible to provide a basic level of care without other clinical support, the limitations of what can be achieved should be recognised. The support of a trained veterinary nurse makes a substantial difference to the level of care and the speed with which emergency patients can be treated. Ideally, both the veterinary surgeons and nurses seeing emergency patients should have shown an interest and aptitude for this patient group; some individuals thrive on the challenging and variable nature of the work whereas others will always find it stressful even with appropriate training and support. Many opportunities now exist to undertake continued professional development in the field of ECC for both veterinary surgeons and nurses and gain further qualifications at both certificate and diploma level; practices are encouraged to support their staff in achieving these goals (see the EVECCS website - Figure 1).

Figure 1. Visit the European Emergency and Critical Care Society website: www.eveccs.org.





For effective functioning of the team it is essential that all individuals know their role and that tasks are delegated appropriately. The veterinary surgeon should focus on tasks that only he as a veterinary surgeon is able to do. This is predominantly making a diagnosis and formulating a treatment plan as well as performing certain invasive procedures. The veterinary surgeon is also required to do much of the communication with the owner particularly at the time of admission when the owner and veterinary surgeon are negotiating and agreeing a diagnostic and treatment plan. The veterinary nurse is able to do a wide range of tasks including patient assessment and monitoring, many diagnostic tests (for example, blood smear evaluation and obtaining radiographs) and many practical tasks such as intravenous catheter placement (depending on the local regulatory situation). By allowing each individual to focus on their role, the efficiency of the team is maximised and patient care is optimised; furthermore allowing individuals to use all their skills promotes job satisfaction. If a trained veterinary nurse is not part of the team then lay members of staff or even owners may need to help (for example, with restraint of patients during procedures) and the veterinary surgeon needs to undertake all clinical tasks; whilst in some circumstances this may be necessary, it is not ideal. All practices seeing emergencies are encouraged to develop a highly trained and skilled nursing team.

As emergency patients can present at any time and successful treatment may take hours to days, it is absolutely essential that each practice has an option for 24-hour care recognising this may mean onward referral once initial stabilisation is complete. If this is not possible, then practices should have processes in place for overnight nursing care and veterinary support. Unfortunately, the abrupt change in disease status that defines the word critical can occur at any time and, when and if it does occur, the period over which we can then intervene in a positive manner to reduce morbidity or mortality may be fairly brief. One of the most important points in addressing what is necessary for effective critical care is thus a recognition that a critical care unit must be able to operate 24 hours a day, seven days a week.

All staff involved with emergencies/critically ill animals should be keen and prepared for the added emotional stress it can entail. Experience is also vital, however, even inexperienced staff who have good attention to detail and the ability to note subtle changes in a patient can be an integral part of the team and their thoughts should not be underrated. Where possible, it may be beneficial to have one nurse to one critical patient, thus increasing the bond and likelihood of noting subtle changes more promptly.

2/ Basic skills and training

All of the team who may be involved with emergencies should be trained in the basic principles of emergency medicine including

effective triage and stabilisation of the major body systems. Triage is the process of rapidly classifying patients on the basis of their clinical priority allowing identification of those patients that need urgent lifesaving help and ensuring that this occurs immediately and before patients with less severe problems are dealt with. Having a standardised approach to triage helps to ensure that nothing is missed during the initial assessment phase and helps to reduce the stress associated with seeing emergencies. Regular feedback on performance and encouragement to develop skills should also be provided especially for more junior members of staff.

Reception or nursing staff are usually the people who have the first contact with owners often by telephone. Understandably owners may be extremely worried at this time. It is extremely important that staff remain calm and reassuring whilst trying to obtain the information needed to judge if the patient should be seen immediately. This can occasionally prove difficult, as the owner's stress may be felt by the person taking the call and obtaining accurate information in a logical way can be challenging. To ensure all relevant information is obtained, it can be a good idea to have a series of question prompts to be used by staff answering emergency phone calls. If initial contact is by telephone, the main aim of the conversation is to ascertain whether the patient has a life-threatening problem. If a life-threatening situation is thought to be present, the patient should be transported to the hospital/practice as soon possible - owner-administered first aid is rarely useful in this scenario and often only delays the patient's arrival at the clinic. Emergencies where veterinary attention should be sought immediately are shown in Figure 2. Once it is known that a patient is coming to the clinic, it is vital that all members of the veterinary team be notified of the estimated time of arrival and be given an idea of the nature of the problem.

Figure 2. Emergencies that should be seen immediately.

- Respiratory distress
- Severe bleeding
- Collapse/unconsciousness
- · Rapid and progressive abdominal distension
- Inability to urinate
- Sudden onset of severe neurological abnormalities
- Protracted vomiting
- Severe diarrhoea
- Witness ingestion of toxin
- · Severe weakness or inability to stand
- Severe pain



All veterinary patients presenting as an emergency should be triaged within 5-10 minutes of arrival at the practice. Veterinary nurses as well as surgeons should be trained in triage and should work as a team such that they can focus their attention on the patients that need them the most. The process of triage involves synthesising information from the patient's history and initial clinical examination, especially an assessment of their major body systems.

Severe life-threatening emergencies are those that involve significant disturbances in the major body systems where there is the potential for rapid deterioration and death. The list of minor emergencies is long but includes problems such as minor wounds, mild vomiting or diarrhoea, polydipsia, "ain't doing right", skin lesions and weight bearing lameness. Although these animals may be presented as emergencies by their owners, the triage process allows their stability to be identified; their full evaluation and treatment can then be delayed until after those patients with life-threatening emergencies have been addressed.

The three major body systems are considered to be:

- Cardiovascular
- Respiratory
- Neurological

When triaging a patient, these systems should always be examined first regardless of any other injuries. A checklist summarising the key parameters evaluated during triage is shown in **Figure 3.** Examination of these systems is a priority as dysfunction in any of these systems is potentially life-threatening. If a patient dies, it is always the result of failure of one of these systems. Although other injuries may be more obvious, they are very unlikely to kill the patient unless they have a secondary effect on one of the major body systems. For example, consider the dog which has been hit by a car and has a fracture of the femur with a large open wound. Although this injury may appear very dramatic, it will not lead to the dog's death by itself. However the haemorrhage from the fracture site may lead to hypovolaemic shock, cardiovascular system compromise and death. The shock will be detected by examination of the cardiovascular system. Thus, the major body systems assessment provides a means of assessing whether the patient's injuries are life-threatening. All parameters should be recorded at the time they are measured. More detail on the assessment of patients for shock (cardiovascular instability) and dyspnoea (respiratory instability) can be found in later chapters in this magazine.

Following on from the triage examination, urgent empirical stabilisation may be started, for example, oxygen and/or fluid therapy. Further in-house diagnostic tests are also commonly performed. In emergency medicine, the focus is on tests that can be performed rapidly and with minimal stress to the patient, and that give information that will

Figure 3. Triage checklist.

- Heart rate
- Mucous membrane colour
- · Capillary refill time
- Pulse quality
- Respiratory rate
- Respiratory effort
- Thoracic auscultation (cardiac and pulmonary)
- Mentation
- · Ambulatory status

help to identify and characterise life-threatening disease processes. Common tests performed are described below:

Minimum database

The MDB is a panel of tests that can be performed from a very small volume (2 microhaematocrit tubes) of blood. It typically comprises the packed cell volume (PCV), total solids (TS), blood glucose and blood urea nitrogen (BUN). Interpreted together the PCV and TS provide a wealth of information about the patient's oxygen carrying capacity and vascular volume status. Blood glucose can be used to identify lifethreatening hypoglycaemia and the BUN can give an early indication that there is an issue with the kidneys or urinary system.

Acid-base, electrolyte and metabolite panel

This ideally includes sodium, potassium, chloride, ionised calcium and lactate as well as measures of oxygen, carbon dioxide and pH. A wide range of life-threatening abnormalities, some of which may need urgent treatment can be identified. Furthermore, problems identified here can provide the basis for a refined differential diagnosis list and help the clinician to prioritise other tests in the overall diagnostic plan. It also acts as a baseline against which further changes can be judged.

Kennel side ultrasound techniques

The use of ultrasound at the "bed side" is increasingly important in emergency work (Figure 4). The A-FAST scan where the ultrasound is used to look for free fluid in the abdominal cavity has been adapted from human medicine and is now well established in veterinary ECC. Increasingly, ultrasound is also being used to evaluate the thoracic cavity with the T-FAST approach for pleural and pericardial space disease and vetBLUE® for assessment of the lung tissue being advocated. Although these techniques do not replace thoracic radiography, they provide a more rapid and less stressful route to obtain information on thoracic pathology in unstable patients.

Throughout this initial assessment process it is vital that all parameters are recorded in the clinical record in a standardised and detailed way.



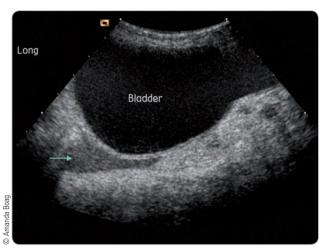


Figure 4. Ultrasound image showing the bladder (labelled) and a small volume of echogenic free abdominal fluid (arrow).

Once these initial tests have been performed and the diagnostic and treatment plan initiated, it is also vital that a monitoring plan is put in place. This should include clear direction on how often to monitor abnormalities that have been identified and actions to take if they are abnormal.

Training is also required in other areas, for example, management of intravenous catheters and other tubes. Hospital-acquired infection (HAIs) can be an important and devastating problem in the critically ill patient, and practices should have clear policies to reduce the risk of HAI. This should include training on hand washing and barrier nursing as well as regular audits of HAIs with feedback to staff. Opportunities for hand washing are based on the WHO's recommendations for hand washing in human healthcare. See a free veterinary adaptation on page 13. Regular morbidity and mortality rounds reviewing cases in a "no blame" way should also be part of the clinical culture. These should focus on patients that experienced an adverse or "near-miss" event and should include a list of actions agreed on to reduce the risk of a similar incident occurring again

3/ Communication

Emergency patients may require 24-hour care and it is clear that a team of people will be involved in caring for the critically ill patient. As discussed above, this team usually consists of a combination of veterinary surgeons and nursing staff. In larger hospitals, junior veterinary surgeons and trainee nursing staff may also be involved. If less experienced team members are part of delivering care, it is vital that there is a clear route for them to seek help and support from a more experienced member of the team should they need it. For critical

care to be effective, good communication between staff is vital. The whole team must be aware of anticipated/possible problems and what and when further interventions (therapeutic or diagnostic) should be performed. Although some patients do deteriorate rapidly, in many cases, careful monitoring and consequently early recognition that a problem is occurring, allows appropriate and potentially lifesaving steps to be taken. Pre-emptive or early action is always better than treating a patient who has already deteriorated significantly. The importance of noting detailed information cannot be over-emphasised. Each time a patient is dealt with, notes should be made on its record. This will build a picture of the patient over the course of its hospitalisation. This may require a different kennel record be used to that in other parts of the practice.

Communication with the owners is also crucial to a successful outcome. Owners are often distressed and very worried about their pet at the time of initial examination. They are also likely to be meeting the veterinary team for the first time. They often have difficult decisions to make about the level of care they want for their animal and their ability to support the associated costs. It is crucial that trust is established rapidly between the client and the clinical team. It is strongly recommended that all veterinary surgeons and nurses working in emergency undergo training in communication skills and receive regular feedback and support in developing these skills in the same way as they do clinical skills.

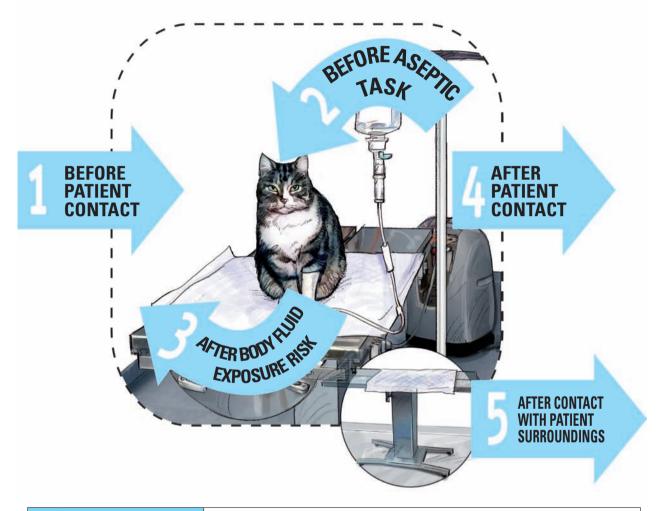
4/ Physical environment and equipment

All practices should have a designated area where emergencies are taken. This room should be easily accessible from as many areas of the building as possible, but not be the main thoroughfare for the practice. It should be staffed with personnel with a keen interest in dealing with the emergency patient. This type of working environment can be extremely hectic and stressful and may not be enjoyed by all members of staff. An efficient emergency room relies on good teamwork, well-trained staff with the skills to pre-empt situations and who have the ability to pick up subtle changes when nursing the critical patient. Early recognition and identification of problems is likely to result in a better outcome for the patient. Ideally, the room should be large enough to provide kennel space so more than one patient can be stabilized at once.

The location of the room is important. Although it is crucial that it is accessible, it should also be able to provide a quiet and calm working environment for staff. The room should be well organized with equipment in the same place all the time, *i.e.*, standard location for the crash box/clippers/catheters. This is more likely to happen if a



Your 5 moments for HAND HYGIENE



1 BEFOR	RE PATIENT ACT	WHEN? Clean your hands before touching a patient when approaching him or her WHY? To protect the patient against harmful germs carried on your hands	
2 BEFOR	RE AN FIC TASK	WHEN? Clean your hands immediately before any aseptic task WHY? To protect the patient against harmful germs, including the patient's own germs, entering his or her body	
	BODY FLUID Sure risk	WHEN? Clean your hands immediately after an exposure risk to body fluids (and after glove removal) WHY? To protect yourself and the healthcare environment from harmful patient germs	
A AFTER CONTA	PATIENT ACT	WHEN? Clean your hands after touching a patient and his or her immediate surroundings when leaving WHY? To protect yourself and the healthcare environment from harmful patient germs	
WITH	CONTACT PATIENT DUNDINGS	WHEN? Clean your hands after touching any object or furniture in the patient's immediate surroundings, when leaving — even without touching the patient WHY? To protect yourself and the healthcare environment from harmful patient germs	

Table 1. Equipment for an emergency room.

	Basic	Advanced
Laboratory	 Centrifuge Refractometer Microhaematocrit tubes Glucometer BUN strip Microscope Equipment for staining blood smears 	Electrolyte/blood gasLactateClottingBiochemistryHaematology
Imaging	Ultrasound capable of detecting free fluid X-ray	Ultrasound suitable for more thorough exam CT
Monitoring	Pulse oximeter Blood pressure	• Multi-parameter monitor with ECG, NIBP and ET CO ₂
Therapeutics	 Oxygen and ability to deliver it Crystalloid fluids (isotonic replacement) Mannitol or hypertonic saline Diazepam Glucose Ca gluconate Furosemide Opioid analgesia Lidocaine Adrenaline Oxygen and ability to deliver it IV antibiotic IV corticosteroid IV sedative/GA induction dru Peripheral IV catheter Urinary catheters Endotracheal tubes Ambu bags Thoracocentesis kit (see Chapter 4) Scalpel blade and suture material 	Colloid fluid Blood products Chest drains Tracheostomy tubes Central lines Drip pump/syringe driver Suction Emergency surgical kit
Other	 Good light source Treatment table Clippers Crash box Laryngoscope Tape for securing catheters Syringes and needles Emergency drug dosage chart Record keeping sheets Telephone 	• Crash alarm

CT = computed tomography; ECG = electrocardiogram; NIBP = non-invasive blood pressure; ET CO_2 = end tidal CO_2

specific member of staff is in charge of the crash equipment including checking maintenance and stock levels.

Equipment and drugs recommended for an emergency room are shown in **Table 1**. It is vital to remember though that equipment is only as good as the people using it and there should be a real focus in the practice on investing in training people to use the equipment well rather than continuously buying new tools.

The room should also be thoroughly cleaned regularly. Implementation of a cleaning schedule and a clear record of who is responsible for

cleaning the area can help with this. Regular cleaning is vital to minimise the risk of hospital-acquired infection (HAI) as critically ill patients are particularly prone to the development of HAIs. All critically ill patients should have their kennels cleaned out at least once a day and clean water and bedding provided, ideally Vet-bed® due to its hydrophobic qualities. Recumbent patients may benefit from mattresses. For canine patients, if stable enough, they may benefit from regular trips outside – the power of sunshine/fresh air should not be underestimated. Good critical care nursing draws on the nurse's ability to provide for the patient's holistic nursing needs. The emergency area is typically used for both dogs and cats and whilst it is not



practical to separate the two species entirely, the team should be aware of the potential stress to cats of being housed close to dogs and should take all opportunities to minimise interaction.

The emergency room should always be ready for the impending emergency, fully stocked with emergency equipment, consent forms and kennel sheets available for immediate use.

Conclusion

All practices will see emergency patients; with a little bit of planning and a focus on appropriate training of the veterinary team. These cases can be treated to a very high standard and be hugely rewarding for the practice to see.



3. Shock and fluid therapy

> SUMMARY

Many patients admitted as an emergency or for intensive treatment present with evidence of circulatory shock (hypoperfusion). The veterinarian must be able to quickly assess the patient so as to determine how serious the hypoperfusion is and start stabilisation, while other investigations are conducted. The initial assessment of a patient with shock must be rapid and is primarily based on an evaluation of the cardiovascular system. Hypovolaemic and distributive shock are the two most common shock conditions in small animals. Treatment for these two types of shock is based on replacing the circulating volume and improving the perfusion parameters using fluid therapy. Isotonic crystalloid fluids are often used as the first choice in treating hypovolaemia and distributive shock. Dosages and administration procedures will depend on how serious the hypoperfusion is.

1/ Shock

Milly is a domestic shorthair neutered female cat, 6 years old. The owner reports that for the last few days, Milly has been eating less and drinking more, and that in the last 24 hours she has become anorexic. In the last 24 hours, she has begun vomiting, with even water being immediately brought back. The owner also reported that Milly had been polyuric over the last few weeks.

During the initial triage, Milly was depressed but responsive. Heart rate of 130 beats a minute, peripheral pulse weak, pale oral mucous membranes and prolonged capillary refill time (Figure 1). No abnormalities were found on auscultating the heart. The jugular veins were not distended.

The respiratory rate is 36 per minute with shallow breaths. Pulmonary auscultation did not identify any abnormal pulmonary sounds. Milly responds with pain to the palpation of the cranial abdomen. Body temperature: 36.7°C.

- What do you think regarding Milly's cardiovascular examination?
- Is Milly in shock? Which type of shock do you think is most probable?
- Which therapy would you chose to stabilise Milly?
- How do you assess her response to treatment?

A) What is shock?

The term "shock" refers to a clinical syndrome rather than a specific pathology. Shock is defined as a state of circulatory insufficiency and tissue perfusion to the extent that oxygen delivery does not meet the patient's requirements. The tissues and cells are exposed to a reduced or inadequate concentration of oxygen and nutrients, such that their metabolism is altered completely. It is important to remember that if tissue hypoperfusion is not treated quickly, it can result in organ malfunction and failure in the organ itself (multi-organ failure or MOF), and the death of the patient.

B) Classification of shock

Circulatory shock is the most common form of shock in our animals and is further classified into four types according to the principal cause. This distinction is very important, because the various forms of shock may need different treatment protocols.

Shock is generally classified as shown in **Table 1.** The two most common forms of shock in our patients are hypovolaemic and distributive shock.





Figure 1. Milly on presentation receiving oxygen supplementation and fluid therapy. Her body temperature is controlled with the use of a hot air heating device (Bair Hugger).

C) Shock and assessment of the cardiovascular system

A meticulous examination of the cardiovascular system provides vital information, not only regarding the presence of primary cardiac problems, but also on the perfusion status.

An examination of the cardiovascular system must include an evaluation of the following:

- Heart rate and heart rhythm
- Quality of the peripheral pulse
- Correlation between the palpation of the pulse and cardiac auscultation
- Colour of the mucous membranes
- Capillary refill time

- Cardiac auscultation
- · Distension of jugular veins

Mentation and temperature difference between the core and periphery may also be of use but are more subjective.

The rapid recognition of shock in an animal is essential to ensure the patient is promptly stabilised. This is why it is important to remember that shock presents differently in cats and dogs.

D) Hypovolaemic and distributive shock in dogs

In a situation of hypovolaemic shock without complications in dogs, the cardiovascular parameters vary according to the severity of the shock, as shown in **Table 2.** The jugular veins are not distended on inspection.

Distributive shock in dogs

The patient presenting with distributive shock may initially have an adequate or only slightly reduced circulating volume; however, this volume is "maldistributed" owing to the acute peripheral vasodilation caused by inflammatory cytokines or bacterial toxins. This leads to a "relative hypovolaemia". Patients in distributive shock can present in a hyperdynamic phase (tachycardia, high pulse rate and narrow "hyperdynamic" pulse quality, hyperaemic mucous membranes with a rapid capillary refill time) or in the hypodynamic phase of shock (tachycardia, weak and faint pulse quality, red congested mucous membranes and prolonged capillary refill time). The colour of the mucous membranes is what principally distinguishes a dog in hypovolaemic or cardiogenic shock from a patient in distributive shock (Figure 2).

E) Hypovolaemic and distributive shock in cats

An evaluation of hypoperfusion secondary to hypovolaemic or

Table 1. Classification of shock.

	Hypoperfusion categories	Hypovolaemic	Distributive	Cardiogenic	Obstructive
Hypoperfusion aetiology Inadequate intravascular volume		Maldistribution of intravascular volume resulting from peripheral vasodilation	Intrinsic failure of heart "pump"	Lower stroke volume as a result of an obstruction to the cardiac output	
	Common causes	Haemorrhaging Vomiting and diarrhoea Loss into third space	Sepsis SIRS Anaphylaxis	Cardiomyopathies Severe valvular diseases Severe arrhythmia	Pericardial effusion Pulmonary embolism



Table 2. Cardiovascular evaluation of uncomplicated hypovolaemic shock in dogs.

	Mild and compensated shock	Moderate shock	Severe and uncompensated shock
Heart rate	130-150	150-170	170-220
Colour of the mucous membranes	Normal	Pale/pink	Very pale, grey
Capillary filling time	Rapid < 1 s	Almost normal < 1.5 s	Slow (> 2 s) or absent
Amplitude of pulse	Increased	Moderate reduction	Reduced
Pulse duration	Slightly less	Moderately less	Reduced

distributive shock in cats is slightly more complicated than in dogs. The oral mucous membranes in cats are generally paler, and although possible, their size makes it more difficult to ascertain a change in the peripheral pulse profile.

A cat in shock often presents in a hypodynamic and decompensated state, characterised by inappropriate bradycardia (heart rate < 140 bpm), hypotension (SAP < 90 mmHg) and hypothermia (< 35° C) (**Figure 3**).

The oral mucous membranes in cats become grey or pale and the capillary refill time is significantly prolonged. Bradycardia and peripheral vasoconstriction contribute to developing hypothermia, which in turn worsens the bradycardia and hypotension. The reasons why cats develop an inappropriate bradycardia are not yet fully understood. The theories offered include: stimulation of the para-sympathetic system,

in addition to the sympathetic system involved in the compensatory response to the shock, responsible for a cardiac slowing effect; diminished responsiveness in the cathecholaminergic receptors in the hypothermic patient; early onset of myocardial dysfunction.

Because the clinical signs of hyperdynamic shock are often not present in cats, the systemic inflammatory response syndrome (SIRS) often associated with distributive shock is suspected and recognised in cats, based on the following criteria: 3 or more of these criteria must exist:

- Rectal temperature > 39.7°C or < 37.8°C
- Heart rate > 225 beats per minute or < 140 bpm
- Respiratory rate > 40 breaths per minute
- White blood cell count (WBC) >19,500 cells/ μ L or < 5,000 cells/ μ L or an increase in the immature forms in circulation by more than 5%

Figure 2. Dog in distributive shock: note the red congested mucous membranes.









Figure 3. Cat in shock receiving initial fluid resuscitation.

In response to the questions for the initial case, Milly suffers from moderate shock, characterised by depressed mentation, inappropriate bradycardia, pale oral mucous membranes and hypothermia. We are most probably recognising hypovolaemic shock with a loss of fluids by way of polyuria and vomiting.

We decide to place a peripheral intravenous catheter in Milly and begin fluid resuscitation treatment. In the meantime, we decide to slowly warm Milly by wrapping her in a blanket and using the Bair Hugger (see box "Approach to hypothermia in shock").

2/ Resuscitation fluid therapy

Fluid therapy must be considered as an essential part of managing emergency and hospitalised patients. Emergency fluid therapy is an essential element in treating acute losses from the intravascular compartment (hypoperfusion secondary to hypovolaemic and distributive shock), and can be used to restabilise and maintain the balance of water, electrolytes and base acid.

Fluid therapy must be considered as a "pharmacological treatment", and similar to other treatments that we use in practice, its effects must be monitored to assess the benefits and the possible complications associated with this (over-hydration). There are no set fluid therapy protocols or ready-made prescriptions according to the patient's pathology or perfusion status; the treatment plan must constantly be changed according to the animal's clinical conditions and requirements.

The objectives of emergency fluid therapy are to:

- Stabilise the effective circulating volume
- · Stabilise the perfusion of organs and tissues
- Stabilise arterial pressure

A) Administering fluid therapy to patients in shock

Please note/warning: Administration of fluids to patients in shock must only be done intravenously and never subcutaneously.

The subcutaneous route must never be used for the following reasons:

- The peripheral vasoconstriction, as a compensatory response to shock, prevents fluids administered subcutaneously from becoming absorbed.
- It is not possible to administer significant volumes of fluids.
- The fluids do not quickly reach the vascular compartment where they are needed.

The placing of one or several peripheral catheters in larger animals allows for large quantities of fluid to be administered rapidly. In newborn or smaller patients, the intraosseous method may be used as an alternative (22-23 gauge hypodermic needles in the humerus or femur).

B) Fluids available

Various fluids are available for treating shock (Figure 4):

- Isotonic replacement crystalloid fluids (NaCl 0.9%, Ringer's solution, Hartmann's solution)
- Hypertonic crystalloid fluids (NaCl at various range: 4.5-10%)
- · Colloid solutions

Please note/warning: as you will see, hypotonic solutions are not mentioned. Hypotonic solutions (*i.e.*, lower tonicity compared to plasma) such as 0.45% NaCl plus glucose 2,5% or 0.18% NaCl plus 4% glucose solution should never be used as fluids in managing hypovolaemia. In addition to not creating an adequate expansion in the circulating volume, they can lead to a rapid reduction in plasma osmolarity and cause serious alterations to the plasma sodium (acute hyponatraemia), leading to the development of serious neurological complications.

C) Isotonic crystalloid solutions

Isotonic crystalloid solution have a similar composition to extracellular fluids. They are the fluids most often used during resuscitation



from shock because they are affordable and can be found in any clinic. All crystalloid fluids will redistribute into interstitial space with only a proportion remaining in the intravascular space: only 20-40% of the fluid administered will persist in the intravascular space after one hour. This characteristic makes crystalloid solutions ideal for using in patients that are hypovolaemic and dehydrated. Careful monitoring is required to ensure the expansion in intravascular volume is sufficient and maintained.

D) Crystalloid solution doses

Books often refer to the so-called shock doses for crystalloid solutions, which in dogs are 60-80 mL/kg and in cats 40-60 mL/kg. Currently, we do not think of resuscitation fluid therapy in terms of administering large quantities of fluid, but rather using a dose (bolus) of fluid according to the needs of the individual patient and severity of the hypoperfusion.

The bolus is generally administered over 15-30 minutes, and not over an hour as recommended in some texts (**Figure 5**).

For dogs, depending on the severity of shock, we may administer:

- 10-20 mL/kg for compensated shock
- 20-40 mL/kg for moderate shock
- 40-60 mL/kg for severe and decompensated shock

Resuscitation fluid therapy must be carried out differently in cats compared to dogs. Cats are not able to tolerate the administration of a large bolus of fluids like dogs. It is easier to cause pulmonary oedema in a cat, secondary to the administration of a high volume of fluids, than it is to cause over-hydration in dogs. This is why we must be very cautious when administering fluid therapy in cats. We need to monitor not only the cardiovascular parameters regularly and frequently, but also the respiratory rate and effort.

In cats, we therefore prefer intermittent administration of small volumes of isotonic crystalloid solutions: 10-20 mL/kg in 15-30 minutes regardless of the severity of the hypoperfusion. The bolus can be repeated immediately if necessary, until the perfusion parameters have normalised. In cats, it is possible to use a 50 CC syringe and manually administer the fluid bolus, so as to better control the quantity and speed of administration.

In hypothermic patients, we must remember that hypothermia reduces the capillary response to the administration of fluids. It is therefore important to try and slowly normalise the body temperature before aggressively stabilising the animal.



Figure. 4. Different types of fluid available for resuscitation.

Figure 5. Pressure bag. In medium to large dogs, when fluids have to be administered rapidly a pressure bag can be used.





Animals that do not adequately respond to the administration of isotonic crystalloid solutions must be reassessed from a clinical perspective. Additional solutions that can be used are colloid solutions and hypertonic saline solution.

E) Hypertonic saline solution

Hypertonic saline solutions (4.5% to 10% NaCl) draw water out of the interstitial and intracellular spaces due to their high tonicity compared to plasma tonicity. For this reason, they should never be used in animals with shock that are also suffering from dehydration.

There are two major indications for using hypertonic saline solution:

- Resuscitation of patients with hypovolaemic shock, which are also suffering from traumatic brain injury
- Resuscitation in larger patients, when it is not possible to administer isotonic crystalloid solutions quickly enough to restore the circulating volume in a suitable timeframe

It is important not to exceed the dose of 2-4 mL/kg for cats and 4-6 mL/kg for dogs, administered over 20 minutes.

F) Colloid solutions

Synthetic colloid solutions (hydroxyethyl starch colloidal solutions (HES) at 6% and 10% available in Europe) contain differently sized macromolecules that do not easily pass through the vascular membrane; they exert a colloid osmotic pressure, thus helping to retain



Approach to hypothermia in shock

Hypothermia (< 35°C), especially in cats, can significantly reduce the cardiovascular response to fluid resuscitation. It is important to slowly and progressively warm up the hypothermic patient, while he/she receives initial fluid therapy resuscitation. It is important not to attempt to quickly bring the body temperature to normal in a hypoperfused patient. A quick increase in body temperature could cause acute peripheral vasodilation, which could worsen tissue perfusion. Initially, one could use blankets to avoid further heat dispersion or hot air heating devices (Bair Hugger).

fluids in the vascular compartment. The colloid particles are usually suspended in 0.9% sodium chloride.

Synthetic colloid solutions cause greater expansion of the vascular volume when compared to isotonic crystalloids, while using a lower volume of administered fluid.

Indications for colloid solutions

Colloid solutions are useful in circumstances where:

- Crystalloid solutions are not enough to stabilise haemodynamics
- When it is necessary to administer a lower quantity of fluids
- In cases of increased vascular permeability

The debate on the safety of colloid solutions

A number of recent studies in human medicine on colloid solutions showed that administering synthetic colloids (HES and dextran) in critical patients is associated with the development of coagulopathies, acute kidney injury (AKI) and a worse outcome. For this reason, use of colloid solutions is now severely restricted in human medicine.

The dilemma in veterinary medicine persists: yes or no to colloids?

There is still no definitive evidence that the use of colloids in canine and feline patients is associated with clinically significant side effects. However, the evidence base is weak with no large-scale studies investigating this. In this regard, indications and dosages on the use of colloids still fall within the guidelines on fluid therapy published by the *JAAHA* in 2013. An awareness of the side effects is recommended.

Dosages for colloidal solutions

Colloid solutions should be used sensibly in patients without alterations in coagulation and without signs of diminished renal function.

The maximum daily dose has not been evaluated in veterinary patients and it is recommended that human guidelines are followed. To summarise, a maximum daily dose of 20 mL/kg for hetastarch product and 50 mL/kg for tetrastarch products should be used. The colloidal solution can be administered in small doses of 5-10 mL/kg over 15-30 minutes.

G) Monitoring patients in shock and fluid therapy

Repeating the physical examination and assessing the cardiovascular parameters is the best way to monitor patients and their response to fluid therapy. A patient must not be considered stable from a circulatory perspective, unless the perfusion parameters have returned to normal or acceptable levels for that patient.



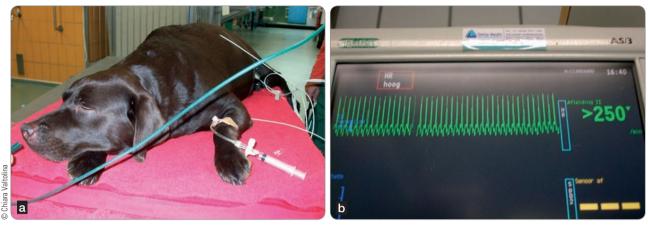


Figure 6. (a and b) Labrador retriever admitted with severe cardiogenic shock, dyspnoea, ascites and supraventricular tachycardia secondary to dilated cardiomyopathy.

We decided to treat Milly's shock by using fluid therapy and chose an isotonic crystalloid fluid like lactated ringer. We administered an initial bolus of 20 mL/kg in 20 minutes. After the first bolus, we noted an improvement in the clinical condition: The pulse had become a little stronger and the heart rate had increased to 160 bpm. We decided to administer another two boluses of 10 mL/kg each over 20 minutes, and at the same time administer a dose of 0.2 mg/kg of methadone to treat the abdominal pain. At the end of the third bolus, the pulse had improved significantly, Milly became responsive and the heart rate was at 190 bpm. Non-invasive blood pressure measured using a Doppler was around 100 mmHg. Milly's shock had resolved, but we continued monitoring Milly by repeating the perfusion parameters' assessment over the next few hours to ensure that the clinical condition did not worsen.

3/ Other types of shock

A) Cardiogenic shock

Cardiogenic shock occurs when the heart's ability to function as a pump and to maintain an adequate cardiac stroke volume for the patient's requirements is compromised. This type of shock arises secondary to mainly cardiac conditions (cardiomyopathies), acute decompensated valvular pathologies or secondary to arrhythmias. Very often, cardiac pathologies can cause cardiogenic shock, along with congestive cardiac failure, with the development of dyspnoea (Figure 6).

Common pathologies in dogs causing cardiogenic shock are dilated cardiomyopathy, rupture of the chorda tendineae or severe degenerative valvular pathologies and severe arrhythmias. Common pathologies in cats include hypertrophic cardiomyopathy and dilated cardiomyopathy. We need to use the information obtained in the medical history and the assessment of the cardiovascular system to make a diagnosis of suspected cardiogenic shock. These patients present tachycardia, often with an irregular rhythm, weak pulses and pale oral mucous membranes, with extended capillary refill time. On cardiac auscultation, heart murmurs or a gallop rhythm are often noted. Jugular veins are often distended and it is possible to discern a jugular pulse. Dyspnoea is often present secondary to pulmonary oedema.

Treatment must focus on improving the perfusion parameters by sensibly using furosemide and a positive inotrope drug (for example, dobutamine, pimobendan, digoxin). Fluid therapy is contraindicated.

B) Obstructive shock

Obstructive shock is perhaps the least common form of shock in veterinary medicine, and can be caused by different pathologies such as pericardial effusion, tension pneumothorax and gastric dilatation and torsion (as a consequence of compression of the caudal vena cava). Characteristic clinical signs include:

- Tachycardia
- Pale mucous membranes
- Prolonged capillary refill time
- Reduced cardiac sounds (muffled or distant) on auscultation
- Dilatation and distension of the jugular veins and pulsus paradoxus

Treatment for obstructive shock consists of removing the obstruction to the venous return (*e.g.*, pericardiocentesis, thoracocentesis or decompression of the stomach in gastric torsion). Fluid therapy may be used but its efficacy is debatable. Furosemide is contraindicated.





4. Approach to the dyspnoeic patient

> SUMMARY

Patients with dyspnoea are among some of the most challenging emergencies. Marked tissue due to oxygen deprivation occurs during dyspnoea and patients suffering from dyspnoea are truly in a critical condition. Small decreases in oxygen intake can be the difference between life and death. Dyspnoea is not always accompanied by a significant increase in frequency of breathing (tachypnoea). Sometimes due to the effort required for breathing, animals cannot concurrently increase the frequency of breathing.

During triage, a patient with dyspnoea must always be regarded as having an acute, life-threatening condition. Minutes count and promptly instituting appropriate stabilisation and therapy is vital. On the other hand, all unnecessary handling should stop, since any additional muscle activity associated with stress or excitement in these patients raises oxygen consumption and can potentially decrease oxygen intake.

The causes of dyspnoea are numerous and diverse. Following initial stabilisation with oxygen, a more targeted therapy requires the clinician to ascertain the location in the respiratory tree from which the dyspnoea originates.

In this chapter, a review of the approach to dyspnoeic patients is provided and important diagnostic and therapeutic considerations are illustrated with the help of case examples.

1/ The dyspnoeic dog

Marly, a one-and-a-half-year-old, neutered female, mixed-breed dog, weighing 11 kg was presented as an emergency after a walk with progressive difficulty in breathing.

As with all emergency patients, an initial triage is carried out according to the ABC approach. During triage, the severity of dypsnoea and with it, the risk of death in the patient is assessed. During assessment of the respiratory system, an examination of the respiratory tract, frequency of breathing, respiratory effort, audible respiratory noise (e.g., stridor), auscultation of the lungs, lung percussion and evaluation of mucous membrane colour is performed.

In Marly, respiration was at a frequency of 24/minute, with marked effort and considerable paradoxical abdominal movement. A distinct stridor was heard upon inspiration. Auscultation and lung percussion were within

normal limits other than the referred noise from the previously mentioned stridor.

Circulatory parameters were unremarkable with a heart rate of 96/min, pale pink mucous membranes, capillary refill time of 1-2 seconds, strong pulses, warm limbs and full consciousness.

A) Emergency therapy

The initial stabilisation of the patient with breathing difficulty should be quick and stress-free. Ensuring a patent airway is essential. Possible foreign material such as mucus should be removed by suction. Any foreign bodies must be removed. Definitive securing of the airways can be done through intubation. However, this is only an option when the patient is in a comatose state or anaesthetised. In the event that an obstruction of the upper airway cannot be removed rapidly, oxygen can be delivered directly into the trachea for a short period through a tracheal cannula. For bypass of the upper airway



due to obstructions or for long-term management, a tracheotomy can be performed.

In every patient with breathing difficulty, oxygen must be supplied to reduce existing or potential hypoxia. There are different ways of supplying oxygen:

- In the simplest cases, this can be delivered by flow-by with an oxygen tube. With this technique, the inspired oxygen concentration can be raised to around 30%. When there is a strong oxygen flow, some animals, especially cats, turn their heads out of the gas flow and then breathe only atmospheric oxygen.
- Administration through an oxygen mask is somewhat more effective. Transparent masks should be used. It should be noted that putting on the mask can lead to stress for some animals who will not tolerate it.
- Administration through an oxygen collar is less stressful. These
 can be prepared from an Elizabethan collar with the front covered
 using plastic wrap (for example, cling film) (Figure 1). However,
 especially in large dogs, humidity and heat can increase within the
 confined space of the collar. Therefore ¼ of the circumference of
 the collar should always be left open.
- With nasal oxygen tubes, the inspiratory oxygen content can be raised up to 60%. Feeding tubes can be placed into the ventral nasal meatus in conscious patients using topical anaesthesia. In cats with dyspnoea particularly, placement of the tubes can be significantly stressful. The oxygen flow with flow-by, mask and tubes should be at around 100-400 mL/kg/min in order to obtain the desired increase in inspired oxygen.
- An oxygen cage offers another possibility for delivering oxygen.
 Here, animals sit mostly stress-free and receive 40-60% of inspired

Figure 1. Dog with self-made oxygen collar from an Elizabethan collar.



oxygen. However, there is short-term oxygen loss upon opening the cage making safe and regular patient examination difficult.

A further, important factor in the stabilisation of patients with dyspnoea is reduction of stress. Dyspnoea is thought to be a very unpleasant experience as in people and dyspnoeic animals are typically very anxious. Hence, they should not be exposed to additional stress, in as much as this is possible. Stress factors can be, for example, excessive handling, placement of intravenous catheters and positioning for radiographic imaging. These measures should only be performed if they are absolutely essential.

Medical treatment can be used to reduce stress, with butorphanol administered by intramuscular injection being a good choice. Other sedative options are described in **Table 1** in the second part of this chapter. This is suitable, above all, for upper airway diseases. For pleural cavity and lung parenchymal diseases, the positive effects of stress reduction must be balanced against a potential reduction in respiratory effort secondary to the sedation.

The initial stabilisation of our patient involved administration of oxygen by mask and subsequently, nasal tube as well as administration of 0.3 mg/kg butorphanol by intramuscular injection. Following sedation, a visual inspection of the oral cavity could be carried out and showed swollen tissue in the caudal part of the pharynx (Figure 2).

During the more detailed clinical examination, hyperthermia was detected with a temperature of 40.6°C. Sedation with butorphanol assisted in reducing temperature. In addition to this, the patient was placed on intravenous fluid therapy with a balanced electrolyte solution.

B) Localisation of the dyspnoea

The next and most important step is the localisation of the dyspnoea. Here, the upper and lower airways, pleural cavity, thoracic wall, lung parenchyma and even non-respiratory causes such as severe anaemia or methemoglobinaemia should be distinguished (see **Table 2**). This is often possible simply on the basis of the clinical examination, but, in some cases, further imaging techniques are necessary. Localisation of the dyspnoea has a great influence on therapy which, if correct, can lead to rapid alleviation of the symptoms.

If present, stridor is generally heard upon first contact with the patient. This usually indicates diseases of the upper airway. The dyspnoea can be further localised to the upper or lower airway according to the respiratory phase in which it is present. Inspiratory dyspnoea is mostly associated with diseases of the upper airway and



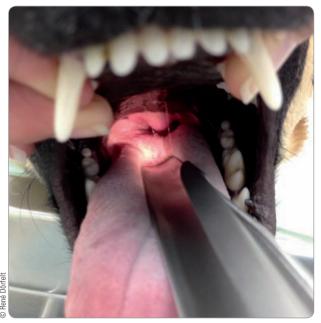


Figure 2. Laryngoscopic view into Marly's pharynx showing swollen tissue.

expiratory dyspnoea with diseases of the lower airway, whilst mixed dyspnoea is associated with lung parenchymal diseases.

The breathing pattern can also be helpful for assessment of the location. Patients without dyspnoea show a synchronous breathing pattern between abdomen and thorax. Asynchronous breathing may occur and is described in the Box below. Abnormal movements of the thorax can occur in chest wall diseases such as rib fractures.

During auscultation, increased lung noise indicates lung parenchymal disease and reduced lung noise indicates pleural cavity disease. The use of percussion makes further differentiation possible, especially in dogs. A drum-like percussion sound is indicative of air accumulation in the pleural cavity (pneumothorax) or in the lungs (e.g., asthma). A dull, muffled percussion sound indicates liquid or tissue, for instance, in pulmonary oedema or haemorrhage. In patients with heart murmurs and breathing difficulty with increased breathing noise, cardiogenic pulmonary oedema is included in the differential diagnosis. Clinical differentiation of the location is difficult in mixed diseases, e.g., pneumothorax in combination with lung haemorrhage (contusion).

Dyspnoea due to non-respiratory causes is not usually associated with significant changes in breathing pattern or auscultation and percussion, but instead with increased breathing frequency and possibly increased breathing effort.



In our mixed-breed dog, a clear inspiratory stridor with a strong abdominal component to respiration was the

principal finding. Auscultation and lung percussion were within normal limits apart from the referred upper airway breathing noise. The breathing difficulty was located to the upper airway.

C) Therapy for breathing difficulty after localisation

For diseases of the upper airway, an attempt must be made to remove the obstruction. In the event this is impossible, an attempt should be made to supply oxygen using a method bypassing the upper airway. This is possible with naso-pharyngeal or naso-tracheal tubes, intubation and, in extreme cases, a tracheotomy. As already mentioned, it often helps to reduce the patient's stress with butorphanol.

Patients with diseases of the lower airway likewise benefit from oxygen therapy, sedation and, if necessary, anti-tussives. In this case, it should be observed that butorphanol possesses a stronger anti-tussive effect than codeine.

For pleural cavity diseases, the foreign material (air or liquid) should be removed from the pleural cavity through thoracocentesis. When pleural cavity disease is due to herniation of the abdominal organs, *e.g.*, in trauma patients with diaphragmatic hernia and severe dyspnoea, urgent surgery may be required. In the case of the stomach being within the thorax, a gastrocentesis can help to reduce the dyspnoea temporarily.

Asynchronous breathing



During normal breathing the chest and abdomen move together in a "synchronous" pattern. With disease, including diaphragmatic rupture, diaphragmatic paralysis, or significantly increased inspiratory effort of any cause, a "paradoxical" or "asynchronous" breathing pattern may be noted. This may include following deviations:

- Inward collapse of the intercostal spaces during inspiration due to negative inspiratory pressure
- Inward collapse of the abdomen during inspiration
- Inward movement of the lower ribs during inspiration due to diaphragmatic contraction
- Inward movement of the ribs during inspiration





Nasal oxygen tube placement

Material:

- Soft urinary catheter or feeding tube
- Marker pen
- Lubricant with local anaesthetic
- Suture material
- Needle holder
- Scissor
- Forceps
- Alternative to suturing tissue glue may be used

Procedure:

- Place some local anaesthetic lubricant at the entrance to the nostril and on the tip of the catheter
- Pre-measure catheter from the tip of the nose to the medial canthus of the eye and mark the catheter with a pen
- · Lift nose tip slightly
- Place the catheter into the nostril and direct it ventro-medially into the ventral nasal meatus
- Feed the catheter carefully forward until the mark is at the tip of the nose



 Secure the catheter in place with, e.g., Chinese finger trap suture technique or glue at the muco-cutaneous border at the nasal fold and with a single suture or glue at the forehead or below the zygomatic arch

In patients with rib factures, a suitable analgesic should be administered, *e.g.*, systemic opioids, or an adequate local anaesthetic in the form of an intercostal block.

For lung parenchymal diseases, the cause should be further differentiated through radiography, ultrasound examination or laboratory diagnostics. In the case of pneumonia, intravenous broadspectrum antibiotics may additionally be administered. In cases of asthma, dyspnoea can be treated with terbutaline 0.01 mg/kg given by intramuscular injection. When cardiogenic pulmonary oedema is suspected, administration of furosemide 2-4 mg/kg in dogs is the best option. Furosemide could also be delivered by CRI at a dose of 0.1 to 0.6 mg/kg/hour.

Non-thoracic diseases must be correspondingly diagnosed and targeted therapeutically following diagnosis.

In our patient, diphenhydramine and prednisolone-21-hydrogen succinate were administered due to suspicion of an allergic reaction. Since this did not lead to improvement, a laryngoscopic examination was carried out under anaesthesia. Here, there was a clear swelling of the larynx with obstruction. To support the patient, a naso-tracheal oxygen tube or tracheostomy could have been placed. The swelling was controlled through local application of phenylephrine. After waking, the patient only breathed with mild stridor. The naso-pharyngeal oxygen tube was left until the next morning. Allergic reactions such as insect bites were questioned as possible causes for this swelling.

D) Further diagnostics

For assessment of the severity and the cause of dyspnoea, a series of further diagnostic measures can be used notably radiography and ultrasound. With radiographic examination, lung parenchyma and the respiratory tract can be visualised in two dimensions starting from the trachea. However, obtaining radiographs examination can lead to stress for the patient due to the precise positioning necessary, as well as representing a certain radiation exposure. Thus, radiographic examinations are only carried out when the patient is stable. Images in two planes are necessary for accurate assessment. In unstable patients, a single dorso-ventral view can be taken to give some approximate guidance although it should be interpreted with caution.

Ultrasound exam is somewhat less stressful for the patient and without radiation exposure. Amongst others, the T-FAST (Thoracic-Focused Assessment with Sonography in Trauma) examination method is available. In this examination, five sites are examined for air and fluid; these sites are the caudal lung fields and over the heart on both sides and in the area of the ventral diaphragm. Changes in cardiac filling and lung parenchyma are also assessed. At the same time, in conditions such as pleural effusion, these can be identified by ultrasound and can then be removed by thoracocentesis.

An arterial blood-gas analysis can be helpful for assessment of dyspnoea. Following initial stabilisation, it is especially helpful for deciding whether a patient needs more oxygen support or whether, in very severe cases, mechanical ventilation is necessary.





Thoracocentesis

Material:

- Clipper
- Disinfectant
- Butterfly catheter for cats and small dogs, hypodermic needle for dogs or venous catheter (16-20 Gauge) or specific over the needle thoracocentesis catheter for all species
- 3-way Stopcock
- Extension line
- 20 cc-50 cc syringes depending on patient size
- · Reservoir for fluids
- Suction device if large amounts of air or fluid are suspected
- Gloves
- Local anaesthetics, for example, lidocaine can be used for analgesia as an intercostal nerve block

Procedure:

- Clip hair between 7th-10th intercostal spaces
- Identify thoracocentesis site at 8th-9th intercostal space
 - Midway between the spine and sternum for cats
 - Dorsal part of the thorax in dogs when air is suspected
 - Ventral part of the thorax if fluid is suspected
- Penetrate the skin with the needle perpendicular to the thoracic wall
- Once through the skin, redirect the needle at a shallow angle either dorsally (if air suspected) or ventrally (if fluid suspected) to penetrate the pleura
- Attach extension line, 3-way Stopcock and syringe to the needle if not done before
- Create negative pressure with the syringe and evacuate the thorax while holding the needle in place





Arterial partial pressures of oxygen (PaO_2) less than 80 mmHg are considered to represent hypoxia whereas those less than 60 mmHg are considered to represent severe hypoxia. If severe hypoxia is present and is not improved by less invasive means of oxygen supplementation, mechanical ventilation should be considered. Mechanical ventilation should also be considered in patients with marked work of breathing even if their PaO_2 is greater than 60 mmHg. This is because over time these patients may develop respiratory muscle fatigue with acute worsening of their oxygenation.

For assessment of dyspnoea patients, pulse oximetry is only occasionally suitable. It is usually impossible to adequately attach a pulse oximeter (for example, on the tongue) with panting dogs or excited patients. Only where reflecting probes are used, *e.g.*, on the base of the tail, can reliable results be obtained. Oxygen saturation

is under 95% for hypoxia and under 90% for a severe hypoxia necessitating oxygen.

2/ The dyspnoeic cat

Introduction

Assessing and stabilising a dyspnoeic cat is considered one of the most difficult tasks an emergency veterinarian has to face. In addition to initially administering oxygen, the approach to the dyspnoeic cat requires a quick assessment of the respiratory pattern and the cardiovascular and respiratory systems so as to localise the origin of



the dyspnoea. Often a mild sedative is necessary to make it possible to carry out certain procedures such as the thoracocentesis and a more detailed physical examination.

A) Initial approach

Priscilla is a four-year-old neutered female Maine Coon cat. On returning home, the owner found her in the garden under a bush, showing visible signs of dyspnoea. The owner immediately brings the cat in to the veterinary clinic where you work.

Past medical history: Priscilla has never had any previous medical problems; she has been regularly vaccinated and wormed. Priscilla is predominantly an indoor cat but she can also get out and come into contact with other cats. The owner reports that perhaps during the last few days, she has been a bit quieter than usual, and perhaps had slightly less appetite. The owner has never heard the cat cough or sneeze.

You begin conducting a quick initial physical examination, while supplementing the cat with oxygen using the flow-by technique. Priscilla is depressed but responsive (Figure 3).

The heart rate is 120 beats per minute, with weak pulses. Mucous membranes are pink/pale and the capillary refill time is more than 1 second; the jugular vein is not distended.



New technology

For patients with heart disease, the plasma concentration of Brain Natriuretic Peptide (BNP) is frequently raised (this is measured as nt-pro-BNP). It has been shown in some studies that the plasma concentration of nt-pro-BNP is greater in cardiac-related dyspnoea compared to non-cardiac dypsnoea. With the help of the nt-pro-BNP level, a conclusion can be drawn regarding the cause of the dyspnoea (identifying or ruling out cardiogenic causes). More recently, so-called "cage-side" SNAP Tests have also been available on the market. The cut-off for cardiogenic dyspnoea is 2,447 pmol/L with a sensitivity of 81% and specificity of 73%.



Figure 3. Priscilla on admission, while she was in the oxygen cage. Note the severe mental depression.

Respiratory rate is 50 breaths per minute with the breathing pattern being rapid and shallow, often with the mouth open.

While continuing with the oxygen flow-by, you decide to auscultate the heart and thorax, without causing any further stress to the patient. During the cardiac auscultation, a systolic 2/6 murmur is found, and thorax auscultation reveals diminished pulmonary sounds ventrally bilaterally and symmetrically. Body temperature is 39°C.

- What is your differential diagnosis?
- What is associated with the ventral reduction in the lung sounds?
- What is your initial approach to Priscilla? What is your priority?
- Which treatment options do you decide on?

Assessing and stabilising a dyspnoeic cat is considered one of the most difficult tasks an emergency veterinarian has to face. A dyspnoeic cat is a very unstable patient that often does not tolerate coercive restraint, without running the risk of worsening the dyspnoea, and possibly causing respiratory arrest.

1) What we shouldn't do

The first rule to remember is that no diagnostic procedure should be undertaken that could place the clinical condition of our patient at significant risk or worsen it. In fact, a dyspnoeic patient could die on the radiology table if we attempted to obtain radiographs as our initial diagnostic plan. Similarly, the dyspnoeic patient could die on the table in the clinic if we tried and restrained it in order to conduct a complete physical examination, to draw blood or place an intravenous catheter.



2) What we should do

So what should we do to treat a dyspnoeic cat? Any cat arriving in the clinic will still be stressed by the trip in the cat carrier. Very often just administering oxygen in the cage or in the carrier and a so-called "hands-off approach" for a while could help considerably to calm the patient and help them to breathe easier. The choice regarding how to administer the oxygen will depend on the equipment available and the least stress it causes to the patient.

These few minutes are useful to obtain some important information from the owner, and to assess the respiratory pattern.

B) Physical examination

A short initial physical examination focusing on assessing the respiratory pattern and the cardiovascular and respiratory systems should be done as soon as the cat is calmer, while still providing supplementary oxygen.

Our task is to quickly localise the dyspnoea, by observing the respiratory pattern and performing auscultation of the thorax, whilst being aware of the most probable differential diagnosis so that the patient can immediately be stabilised (**Table 2**).

Table 1. Sedation dosage.

Drugs	Type of drugs	Administration	Dosage	Desired effects
Morphine	Pure μ-opioid	IM, IV (SC)	0.1 mg/kg	Sedation and vasodilatation of pulmonary veins
Methadone	Pure μ-opioid	IM, IV (SC)	0.1-0.2 mg/kg	Sedation
Butorphanol	Agonist/antagonist opioid	IM, IV (SC)	0.1-0.3 mg/kg	Sedation and anti-tussive effect
Alfaxalone	General neurosteroid anaesthetic, blocking GABAA receptors	IM, IV	1-2 mg/kg	Sedation
Acepromazine	Phenothiazine	IM, IV (SC)	5-20 μg/kg	Sedation for upper airway diseases

Table 2. Localisation of dyspnoea and most common differential diagnoses.

Localisation of dyspnoea	Respiratory pattern	Auscultating the thorax	Differential diagnosis
Upper respiratory tracts	Obstructive dyspnoea Extended initial inspiratory phase with inspiratory effort	Often respiratory stertor or stridor audible without a stethoscope	Acute or chronic rhinitis, nasal or pharyngeal polyps, pharyngeal oedema, laryngeal oedema (inflammation, extraneous bodies or neoplasia), laryngeal paralysis and brachycephalic obstructive airway syndrome
Pulmonary parenchyma	Restrictive dyspnoea. Mixed respiratory effect, both inspiratory and expiratory	Distinctive respiratory sounds and wheezing with smaller and larger bubbles ("crackles")	Cardiogenic pulmonary oedema, neoplasia, pulmonary contusions, verminous pneumonia (<i>Aelurostrongylus abstrusus</i>) and rarely aspiration pneumonia
Lower respiratory tract	Obstructive dyspnoea. Extended initial expiratory phase with expiratory effort. Coughing	Increase in pulmonary sounds with wheezes on auscultation	Chronic bronchial pathologies, asthma
Pleural space	Restrictive dyspnoea. Superficial and quick breathing, often with asynchronous respiration	Diminished pulmonary sounds with auscultation	Pneumothorax, pleural effusion (secondary to left cardiac insufficiency, neoplasia, pyothorax, trauma)



The cardiovascular examination must include an assessment of the perfusion status and cardiac auscultation. Given that congestive heart failure is a common cause of dyspnoea in cats, in our initial examination, we must recognise the clinical signs that could suggest an underlying cardiac pathology (distension of jugular veins, jugular pulse, gallop rhythm, cardiac murmurs). An evaluation of the body temperature is equally important, because pathologies such as congestive heart failure and trauma are often associated with hypothermia in cats.

Priscilla's initial assessment reveals secondary clinical signs of peripheral hypoperfusion (inappropriate bradycardia, pink/pale mucous membranes and prolonged capillary refill time) and restrictive dyspnoea with superficial and frequent respiration, characteristic of pleural space pathology. The presence of fluid in the pleural space is confirmed by auscultating the thorax and decreased pulmonary sounds ventrally.

The cardiac murmur could lead one to initially consider congestive heart failure as the primary pathology for the dyspnoea. However, Priscilla's body temperature is high, and hyperthermia is never associated with congestive heart failure.

C) Use of sedatives to manage dyspnoeic cats

Most dyspnoeic cats benefit from a mild sedative. It is worth remembering that the stress associated with dyspnoea only worsens the dyspnoea itself. This is why we recommend administering a drug that can reduce the demand for air and the sense of dyspnoea. Due to the fact that, initially, we do not know what the primary cause for the dyspnoea may be, we do not recommend using sedative drugs that could negatively affect the cardiac cycle, like $\alpha 2$ -agonist drugs (medetomidine and dexmedetomidine) or sedative drugs from the phenothiazine family (acepromazine). Recommended drugs for sedating the dyspnoeic patient are pure mu opioid drugs (methadone and morphine) or an opioid agonist/antagonist like butorphanol. Alfaxalone may also be used via intramuscular injection if the previously mentioned drugs do not have sufficient effect. Acepromazine is rarely used in dyspnoeic cats (Table 1).

D) Initial stabilisation of the dyspnoeic

Oxygen therapy and sedation are often the initial treatments that we should offer a dyspnoeic cat. It should be remembered, however, that oxygen therapy is not enough to stabilise dyspnoeic patients with pathologies involving the pleural space. In these cases, thoracocentesis

The medical history of the dyspnoeic cat



A solid medical history of the dyspnoeic cat can provide basic information to better understand the primary pathology initially causing the dyspnoea.

It is important to ask the owner whether the cat has access to the outside, and whether it lives with other cats (and the health status of the other animals). It is important to ask when the clinical signs began and what their progress has been. Keep in mind, however, that it is often difficult for cat owners to notice intolerance to physical exercise when cats lead a predominantly sedentary life.

It is important to enquire whether the cat is coughing, remembering that cats cough especially with bronchial pathologies, and almost never in the case of pulmonary or pleural space pathologies, due to the different distribution of cough receptors compared to dogs.

is essential as a therapeutic and diagnostic procedure. Thoracocentesis in cats can be done using a butterfly, 3-way Stopcock and a 10 or 20 cc syringe (see "Thoracocentesis" Box on page 28). The fluid that is taken must always be assessed cytologically, and a sample should be kept for culture testing.

In emergency situations, the initial physical examination and the information provided with the medical history should be enough to begin treatment to stabilise the patient. There will be situations where the patient is so dyspnoeic that it will not be possible to conduct a physical examination. In these exceptional cases, stabilisation can be attempted using a sedative and by administering a dose of corticosteroids, bronchodilators and furosemide.

In Priscilla's case, the priority is to initially improve the dyspnoea and so the thoracocentesis must take priority over placing an intravenous catheter. On conducting thoracocentesis, purulent fluid is found bilaterally in the thoracic cavity. 80 mL and 60 mL are drained from the left and right hemithorax respectively (Figure 4).





Figure 4. Purulent exudate.

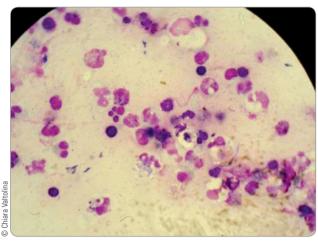


Figure 5. Cytological evaluation of the pleural effusion: note the high cellularity, consistent mainly with degenerated polymorphonucleated cells and macrophages. Some of the inflammatory cells contain intracellular bacteria.



Figure 6. Cat with pyothorax with bilateral chest drains. Via the chest drains, the pleural space is drained and flushed using warm NaCl 0.9% solution (a, b, c and d).



A quick cytological examination of the fluid shows a number of inflammatory toxic cells, as well as intracellular bacteria (Figure 5). Priscilla is suffering from pyothorax and secondary distributive shock.

E) Most common pathologies in dyspnoeic cats and initial stabilisation

1) Congestive heart failure

Congestive heart failure can present in cats of all ages. Hypertrophic cardiomyopathy is the most common cardiac pathology. Affected patients present with acute mixed dyspnoea, associated with pulmonary oedema and/or pleural effusion. On auscultating the thorax, it is possible to discern wheezing with smaller and larger bubbles in the pulmonary fields ("crackles"). Often, diminished pulmonary sounds may be noted ventrally if there is pleural effusion (transudate or modified transudate). Alterations in the peripheral perfusion and hypothermia are characteristics of congestive heart failure.

The emergency treatment for this pathology includes administering oxygen, in addition to administering the loop diuretic furosemide. In cats, the furosemide is administered as an initial intramuscular dose (or possibly intravenous) at 2 mg/kg and then repeated every hour up to 4-5 administrations at 1 mg/kg, once again as an intramuscular dose.

2) Feline "asthma"

Feline "asthma" (now named "feline allergic airway disease") is a pathology affecting the lower respiratory tracts in cats. It can be diagnosed in cats of any age, but more often affects younger patients

and middle-aged adults. Cats with asthma suffer from expiratory dyspnoea, secondary to acute bronchoconstriction and coughing is a common clinical sign. Expiratory pulmonary whistling can be found on auscultating the thorax. Cats affected by asthma are normotensive and normothermic.

Emergency treatment for these patients requires parenteral administration of a bronchodilator [β 2- agonist like terbutaline (0.01 mg/kg IM, SC)] or a phosphodiesterase inhibitor like aminophylline (5-10 mg/kg IM, SC), in addition to a corticosteroid (dexamethasone – 0.1-0.2 mg/kg IM, IV, SC).

3) Pyothorax in cats

Pyothorax in cats is a pathology characterised by an accumulation of a purulent and septic pleural effusion. The pathogenesis of pyothorax in cats seems to be as a result of wounds from a penetrating bite/scratch. The pathogenic organisms involved are *Pasteurella spp.* and anaerobes (*Nocardia* and *Actinomyces spp.*). A cat affected by pyothorax often presents with alterations to the cardiovascular system (distributive shock); hypothermia or hyperthermia may also be found.

The dyspnoea is restrictive when associated with the pleural effusion. Carrying out thoracocentesis is essential to stabilise the patient. Once the dyspnoea has improved, an intravenous catheter is placed so fluid therapy can be administered to correct the hypoperfusion. Broadspectrum antibiotics are administered (amoxicillin and clavulanic acid) pending the result of culture testing. Analgesics may also be administered. The positioning of the bilateral thoracic drains to drain and flush the pleural space in more stable cats forms part of the treatment protocol for these patients (**Figure 6**).



5. The vomiting emergency patient

> SUMMARY

In dogs, one of the most common reasons for presenting to an emergency care practice is vomiting with 12% of cases presenting to a primary care emergency service having vomiting as their primary complaint. In cats, vomiting represents a smaller proportion of emergency presentations but should not be underestimated. Vomiting is a non-specific symptom of multiple disease processes. In many cases, it is self-limiting but, in a proportion, it occurs secondary to severe and potentially life-threatening underlying disease. The skill of the emergency clinician is to identify which patients have self-limiting causes where symptomatic (empirical) therapy is appropriate and which patients require a diagnostic work-up with treatment of the underlying disease. Furthermore, for patients with significant underlying disease it is crucial to work out whether surgery is required to address the problem.

In the following chapter decision-making criteria are studied for various causes of vomiting and therapeutic possibilities are discussed. A case is used throughout to emphasise some of the challenges seen in real patients.

1/ Initial presentation and assessment

A four-year-old, neutered female Weimaraner presented as an emergency for vomiting that had been on-going for seven weeks. The vomiting always took place in the morning before feeding and was mostly bile. An examination along with laboratory work and faecal testing at the referring veterinarian's practice showed no abnormalities. The dog had had recurring episodes of vomiting and diarrhoea since it was adopted at a young age.

The symptoms worsened three days before its current presentation with the dog now vomiting 2-3 times daily. Upon repeat presentation at the local practice, radiographs and ultrasound showed no abnormality aside from a subjectively thickened stomach wall. Bloodwork showed that cPLI was very slightly raised. The dog was treated with pantoprazole, amoxicillin/clavulanic acid, maropitant and Vitamin B12. In the last 12 hours, the vomiting had worsened still further. During this time, the dog had vomited 6 times and seemed weaker than usual.

Clinical examination:

The short clinical examination following initial triage showed no significant abnormalities other than a low heart rate. The findings were as follows:

A. Clear

B. Breath frequency: 32/min

C. Heart rate: 52/min, strong pulses, pink mucous membranes, capillary refill time of 1-2 s

The dog's general condition was stable, it had a rectal temperature of 37.9°C, was not clinically dehydrated and showed no abnormality apart from a cranial abdomen that was moderately painful upon pressure.

List of problems:

- Chronic vomiting with recent clinical deterioration
- Abdomen moderately painful upon pressure

It is important to differentiate self-limiting vomiting from vomiting requiring further work-up. If a non self-limiting cause of vomiting is suspected, the clinician must decide which further tests are warranted and how these should be prioritised. Importantly, patients requiring surgical treatment should be identified at the earliest possible opportunity.



Historical information that suggests the patient should receive further work-up with an attempt to reach a diagnosis include presentation for a second opinion, failure to normalise despite anti-emetic therapy, more than 5 vomiting episodes in the last 12 hours, and pre-treatment with non-steroidal anti-inflammatory drugs (NSAIDs). Furthermore, indications of concurrent medical disease such as polyuria and icterus point to underlying systemic disease. For vomiting in puppies under three months old or toy breeds under six months old, there is a real concern over the development of hypoglycaemia, since these animals typically have insufficient gluconeogenesis and the animal's blood glucose levels should be measured with stabilisation as appropriate.

From the clinical examination, indicators that a thorough work-up should be performed include abnormalities in the triage exam such as shock or tachypnea/dyspnoea reddened or pale mucous membranes, reduced or prolonged capillary refill time, diminished consciousness or dehydration, hypo- or hyperthermia and painful abdomen. Especially in cats, this may present as localised pain rather than a diffusely painful abdomen.

At first, our patient seemed uncomplicated in terms of its history. This was a dog with chronic vomiting. The worsening over recent days, as well as further deterioration despite anti-emetic therapy, seemed suspicious. Although the clinical examination shows no significant abnormality, the painful abdomen stands out. These findings together point to a cause of vomiting requiring further diagnostic work-up.

2/ Selecting diagnostic tests

The causes for vomiting are numerous and diverse. Firstly, gastro-intestinal causes such as gastrointestinal (GI) tract inflammation, distension, ischaemia and obstruction are all possible. Equally, there are extra-gastrointestinal causes such as central nervous system disease, stimulation of the vomiting centre and organ dysfunction, particularly hepatic and renal disease. Laboratory testing as well as imaging of the gastrointestinal tract and abdomen is appropriate for the diagnosis.

In our patient, further diagnostics had already been performed, without any significant findings. Nevertheless, the patient had deteriorated and there was a clear rationale for repeating some of the tests. The patient exhibited a painful abdomen, so the cause of the vomiting was probably due to gastrointestinal disease. Due to the deterioration, a repeated image-based diagnostic approach was logical.

A) Imaging

The decision regarding the kind of imaging used to help reach a diagnosis depends on various factors. Occasionally, radiographs as well as ultrasound examinations are necessary. In the radiographic examination, an overall view of the abdomen can be achieved. Radiopaque foreign bodies, as well as severe gaseous distension of the intestines and free abdominal air can be reliably identified. Moreover, the radiographic images can be reviewed rapidly in-house and sent externally for a second opinion if necessary. However, radiographs do not allow assessment of the organ structure or organ function (e.g., intestinal motility).

Absolute indications for surgery include free air in the abdomen, gastric dilatation volvulus or radiopaque foreign body in the small intestine with signs of obstruction. Surgery should be strongly considered if signs of obstruction are present as evidenced by dilated intestinal loops with a diameter greater than the height of the vertebral body of the 5th lumbar vertebra. Gaseous distension of the small intestine should be distinguished from distension of the caecum and rectum, which may be normal. If the radiographs are not conclusive, an ultrasound examination can produce further information. In a few cases, a contrast radiographic examination of the gastrointestinal tract using barium is necessary.

Ultrasound examination requires a level of skill and experience by the clinician. This method is not only device-dependent but also very operator-dependent. At its simplest a relatively inexpensive machine and a basic level of skill allow detection of free abdominal fluid. With a good quality machine and a skilled operator, free air may be identified as well as free fluid. Gastrointestinal foreign bodies and linear foreign bodies, as well as intussusception, are likewise recognisable with some practice. Assessment of the intestinal wall and intestinal content, as well as of the pancreas, kidneys and other abdominal organs, is possible. Ultrasound guidance may be very helpful in obtaining samples of free abdominal fluid as well as aspiration of abnormal organs.

Ultrasound findings such as foreign bodies in the small intestine and dilated intestinal loops, particularly if these contain ingesta that appear to be moving backwards and forwards, suggest surgery is required. If dilated loops of bowel are identified, it is appropriate to try to follow these intestinal loops with the ultrasound. Frequently, the cause of an obstruction such as a foreign body or intussusception is to be found at the end of the dilated area. Sudden changes in intestinal wall thickness or GI tract lumen should likewise be considered to be a cause of concern. Free abdominal air can also be identified by ultrasound and, unless the patient has had a laparotomy in the preceding few days, is an indication for surgical exploration.



In the case of our Weimaraner, radiographic and ultrasound examinations of the abdomen were carried out. In the radiographic examination (Figure 1), a liquid-filled stomach and reduced serosal detail in the cranial abdomen were identified. Aside from the fluid in the stomach, the ultrasound examination revealed no further abnormalities.

B) Laboratory diagnostics

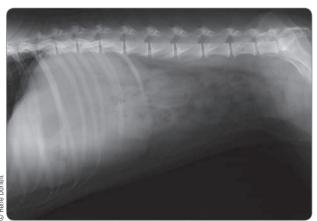
When should a laboratory test take place? Many tests can be justified in vomiting patients, but it is important that the diagnostic plan should be tailored to each patient with a focus on the tests that will help rule in or out the most likely differential diagnoses for that individual. Furthermore, laboratory tests (particularly acid-base and electrolytes) are important to aid patient stabilisation and as such should be performed in the vast majority of patients where an underlying cause for the vomiting is sought.

Laboratory testing is essential for the diagnosis of some extragastrointestinal causes of vomiting such as azotaemia or diabetic ketoacidosis. Further special tests such as cPLI and CRP likewise help the diagnosis to be better identified.

Which parameter should be determined?

The determination of haematocrit and total protein is appropriate for assessment of the hydration level in all patients with significant vomiting. Profuse vomiting can lead to severe dehydration and haemoconcentration. In some diseases such as, for example, proteinlosing enteropathies or septic diseases, the total protein can be reduced without a drop in haematocrit. The leukocyte count and differential may suggest an infectious cause for the disease, for example, in

Figure 1. Radiograph of the vomiting dog in case report in laterolateral view.



Indications for surgery in vomiting patients



- Free air in abdomen
- Septic peritonitis
- Bile peritonitis
- Necrotising pancreatitis
- Foreign body
- Linear foreign body
- Intussusception
- Volvulus
- Gastric dilatation volvulus
- Uroabdomen if urinary tract injury is thought to be extensive

puppies with vomiting. If a neutropaenia is present, parvovirus infection can be suspected.

To rule out renal causes, urea and creatinine levels should be measured. Pre-renal causes of azotaemia are common in vomiting patients and the importance of an azotaemia can be determined by serial measurements of urea and creatinine; the initial values determined serve as a baseline. If urea and creatinine are increased, a pre-renal azotaemia should be ruled out by determining the urine specific gravity. Infectious diseases such as leptospirosis can lead to vomiting and azotaemia, and the likelihood of this disease may be determined by geographical location. If leptospirosis is suspected, further infectious disease testing is warranted.

Venous blood-gas analysis is warranted in the majority of patients to identify abnormalities in acid-base status and guide therapies for this. Blood-gas analysis can also help to better assess disease severity in patients where this is unclear and may point the clinician in the direction of some underlying disease. Animals with severe loss of fluids secondary to vomiting frequently demonstrate a lactic acidosis. Profuse vomiting may also lead to a metabolic alkalosis, and mixed acid-base disturbances are common. Metabolic alkalosis is most frequently (albeit not exclusively) associated with an obstruction in the proximal gastrointestinal tract (pylorus and duodenum). If this abnormality is seen on blood gas analysis, the possibility of Gl obstruction should be strongly considered.

Just as with blood gas analysis, measurement of electrolytes can give clues as to an underlying disease. Hypochloraemia, similarly to metabolic alkalosis, is an indicator of profuse vomiting and GI tract



obstruction. Hyperkalaemia may suggest hypoadrenocorticism, especially in combination with hyponatraemia. Other causes for hyperkalaemia include pre-analytic errors, massive acidosis, anuric/oliguric renal failure and urinary tract obstruction/rupture. As a consequence of fluid loss associated with vomiting, abnormalities of sodium may occur (both hypernatraemia and hyponatraemia may be seen depending on the nature of the lost fluid); hypokalemia is also common. These abnormalities should be treated according to their severity. In patients with severe vomiting, hypocalcaemia can also be seen. This is recognised as a negative prognostic factor in critically ill patients. Ionised calcium should be measured instead of total calcium.

In patients with hypoperfusion or where the severity of cardiovascular impairment is unclear, measurement of lactate can be helpful. Assessment of cardiovascular status may be challenging if a high vagal tone associated with GI disease means the heart rate does not increase as expected in patients with compensated or early decompensated shock. Lactate levels over 2.5 mmol/L indicate a mild and over 5 mmol/L a moderate impairment of perfusion.

In critical patients with clinical indications of sepsis, glucose measurement is important. Patients with inadequate liver function cannot carry out sufficient gluconeogenesis to maintain blood glucose levels. If liver function is considered to be normal, hypoglycaemia can be an indicator of a septic process in vomiting patients. If hypoglycaemia is identified, the cause should be pursued diagnostically in every case. Blood glucose should be regularly monitored. Seizures can occur when glucose levels reduce to 3 mmol/L or less. Severely hypoglycaemic patients should be treated with glucose administration initially as a bolus, followed by glucose-supplementation of their intravenous fluids.

Canine pancreatic lipase immmunoreactivity (cPLI) is increased in patients with pancreatitis. If cPLI is negative, pancreatitis is unlikely. If cPLI is positive, pancreatitis is a strong possibility for the diagnosis; however, it must be remembered that other GI diseases can cause secondary pancreatitis and thus the diagnostic evaluation should not be stopped.

In puppies or young adult dogs without clear vaccination status, vomiting, especially when in combination with diarrhoea, should prompt a test for parvovirus. The faecal SNAP Parvo Test is suitable in emergencies.

In our dog, a venous blood-gas analysis, a haematology panel and a serum biochemistry test were done. There was conspicuous moderate metabolic alkalosis, mild haemoconcentration and a slight increase in alkaline phosphatase (Tables 1, 2 and 3).

Indications for further evaluation of vomiting patients



History

- Profuse vomiting (> 5 times within the last 12 hours)
- · Constant retching
- On-going vomiting despite treatment with anti-emetic
- History of NSAID use
- Haematemesis
- Faecal vomiting
- Other signs of medical disease e.g., PU/PD
- History of toxin ingestion

Clinical examination

- Tachycardia or bradycardia
- Severe salivation
- Reddened or pale mucous membranes
- · Increased or decreased capillary refill time
- Tympanic abdomen
- Hyper- or hypothermia
- Painful abdomen
- · Reduced consciousness

Laboratory

- Haemoconcentration
- Anaemia
- Leucocytosis or leucopenia with left shift (increased proportion of band cells)
- · Increased lactate
- Azotaemia
- Hyperkalaemia
- Hypochloraemia
- Metabolic alkalosis
- Hypoglycaemia
- Hypocalcaemia
- Hypercalcaemia
- Increased cPLI

C) Ascites analysis

In the event free fluid is identified in the abdomen either clinically, on ultrasound or on radiographs, an effort should be made to obtain a sample for subsequent analysis. For large quantities of fluid, a blind abdominocentesis technique can be used by placing a needle into the abdomen in the periumbilical area. If fluid is not obtained, a 4-quadrant technique can be used. Targeted abdominocentesis is possible with



ultrasound guidance. This allows small amounts of fluid to be sampled safely. In the event that there is insufficient fluid to obtain a sample, intravenous fluid therapy should be started, and the abdomen assessed following this. Frequently, ascites increases in volume following fluid therapy, and a sample can be obtained.

The fluid obtained should be examined visually and undergo laboratory analysis. Cloudy fluid is usually indicative of a septic or neoplastic effusion. In septic effusions, the specific gravity and total protein are generally higher than with non-septic effusions (**Table 4**).

Furthermore, the cell count in the abdominal fluid should be determined. With septic effusions frequently more than 7,000-10,000 cells/µL are seen. The cells should be microscopically evaluated on a stained smear (e.g., Diff-Quik). In the event intracellular bacteria are seen, septic peritonitis can be diagnosed. However, it should be noted that, in patients with gastric or proximal duodenal rupture, bacteria might be low in number and hard to identify. The morphology of the leukocytes is also important. Plump, karyolytic cell nuclei indicate inflammatory fluid, whilst over-segmented, karyorrhectic cells appear often in postoperative patients or in ascites that is not associated with significant inflammation. In addition, biochemical analysis can be performed on the ascitic fluid. Glucose, lactate, creatinine and bilirubin may be measured. In patients with abdominal sepsis, glucose is broken down in the fluid by leukocytes and bacteria. Therefore in the patients with septic abdomen, glucose concentration tends to be reduced in comparison to the blood glucose by more than 1.1 mmol/L (20 mg/dL). In patients that have received glucose therapy and in diabetic patients,



Perioperative stabilisation

- Shock infusion and rehydration (aim: improvement of heart rate and lactate to normal or near normal levels)
- Optimisation of blood pressure with fluid therapy and, if necessary, vasoactive medications (aim: mean arterial blood pressure > 80 mmHg)
- Optimisation of venous fluid balance (aim: central venous pressure 5-8 cmH₂O)
- Optimisation of urine production (aim: > 1 mL/kg/h)
- Normalising hypoglycaemia (aim: Glucose > 3.5 mmol/L)
- Normalising severe acid-base disturbances (aim: pH > 7.2)
- · Antibiotic administration if infection is suspected

Table 1. Venous blood-gas analysis of the vomiting dog in the case report.

Parameter	Value	Reference range	Too low Normal		Too high
pН	7.43	7.31-7.43		~	
pCO ₂	51.2 mmHg	32-54		~	
pO_2	40.3 mmHg	30-50		~	
HCO ₃	30.5 mmol/L	19-24			x
BEecf	9.5 mmol/L	-2.5-2.5			×
Na	153.1 mmol/L	146-165		~	
K	3.65 mmol/L	3.5-5.6		~	
Ca++	1.28 mmol/L	1.2-1.4		~	
Cl	106 mmol/L	105-118		~	
Anion gap	17.2 mmol/L	15-20		~	
Glucose	5.2 mmol/L	3.9-6.5		~	

the test is not interpretable. Lactate accumulates in septic fluid as it is produced by leukocytes and bacteria. Therefore in septic peritonitis, lactate is increased by more than 2 mmol/L compared to blood. Unfortunately, the analysis of these parameters does not reliably distinguish the cause of abdominal effusion in patients with intraabdominal neoplasia, in the first days post-operatively, or when drainage tubes are still in the abdomen.

If bile peritonitis is suspected, the bilirubin is measured in the abdominal fluid and in the blood. A bilirubin concentration in the abdominal fluid that exceeds the bilirubin concentration of the blood by more than two indicates bile peritonitis. Uroabdomen behaves similarly. Here, creatinine, potassium and urea are measured. In the event that creatinine in the abdominal fluid is double that in a concurrently obtained serum sample, uroabdomen may be diagnosed. Potassium and urea gradients (i.e., the difference between the value obtained in the abdominal fluid and blood) are also supportive with the abdominal fluid having a level that is 1-1.4 times the serum level. In terms of treatment, patients with bile peritonitis invariably require surgical treatment. For uroabdomen, the likely cause of urinary tract rupture should be assessed; if the rupture is likely to be large, surgery is required. Smaller ruptures may heal with time and urinary diversion should be performed during this time via indwelling urinary bladder catheter and/or abdominal drain.



Table 2. Haematology panel of vomiting dog in the case report.

Parameter	Value	Reference range	Too low	Normal	Too high
WBC	7.25 10º/L	5-16		~	
RBC	9.01 10 ¹² /L	5.5-9.3		~	
HGB	12.9 mmol/L	7.45-12.5			×
НСТ	0.549 l/L	0.35-0.58		~	
MCV	60.9 fL	58-72		~	
МСН	1.432 fmol/L	1-1.4			X
мснс	23.5 mmol/L	19-21			X
PLT	259 10º/L	150-500		~	
NEUT#	2.92 10º/L	3-9	×		
LYMPH#	1.88 10º/L	1-3.6		~	
MONO#	0.96 10º/L	0.04-0.5			X
EO#	1.48 10º/L	0.04-0.6			X
BASO#	0.01 10º/L	0-0.04		~	

Based on the recent worsening of vomiting with repeated vomiting over a short time, a stomach filled with fluid, haemoconcentration and the presence of a metabolic alkalosis, a physical or functional obstruction of the pylorus or proximal small intestine is the prime differential diagnosis. Hence, after initial fluid therapy, the patient was anaesthetised for a gastroduodenoscopy. Due to the gastric distension and the high risk of regurgitation and aspiration, a rapid sequence induction was carried out with rapid securing of the airway through an orotracheal tube. Subsequently, the stomach was emptied using a stomach tube. During endoscopy, a tough, yellow foreign body was found in the proximal duodenum directly distal to the pylorus. This could not be removed by endoscopy and the dog underwent laparotomy; the hard rubber foreign body was removed by retropulsion into the stomach and gastrotomy (Figure 2).

3/ Medical management of vomiting patients

In the event that further diagnostic tests are not warranted or that there is no indication for surgical intervention following diagnostic tests, the management of vomiting patients consists of optimisation

Table3.Ser umpar ametersof they omitingdoginthecas er eport.

Parameter	Value	Reference range	Too low	Normal	Too high
ALT	58 U/L	18-110		~	
AP	180 U/L	13-152			×
Bilirubin- Total	2.5 μmol/L	0-5.26		~	
Protein	72.4 g/L	55.5-77.6		~	
Albumin	38.1 g/L	31.3-43		~	
Urea	3.9 mmol/L	3.52-10.78		~	
Creatinine	83 µmol/L	44-125		~	
Glucose	5 mmol/L	3.79-6.58		~	
P	1.13 mmol/L	0.86-2.01		~	
Cl	105.6 mmol/L	105-118		~	
Na	148.7 mmol/L	139-163		~	
K	4.12 mmol/L	3.8-5.5		~	
Ca	2.73 mmol/L	2.2-2.8		~	

Table 4. Differentiation criteria between transudate, modified transudate and exudate (according to Silverstein and Hopper, *Small Animal Critical Care Medicine*, 2009).

Effusion	Total protein	Specific gravity	Cell count
Transudate	< 25 g/L	< 1017	< 1,000/μL
Modified transudate	25-50 g/L	1017-1025	500-10,000/μL
Exudate	> 30 g/L	> 1025	> 5,000/µL

of perfusion, management of secondary electrolyte and acid-base disturbances, anti-emetic therapy, gastrointestinal protectants, analgesics if necessary, and adequate nutrition. If a specific medical cause of vomiting is identified, then this should also be treated. The responsible veterinarian must decide whether the treatment must be done in-hospital or if the patient can be treated as an outpatient.

Indicators for in-patient care are:

- Hypoperfusion
- Dehydration greater than 8%



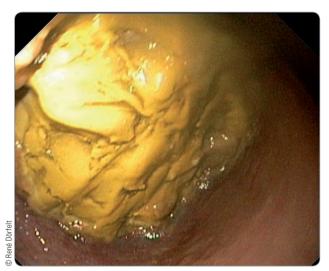


Figure 2. Endoscopic image of foreign body of the vomiting dog in the case report.

- Severe acid-base disturbances
- Hyperkalaemia
- Hyperlactataemia
- Moderate to severely painful abdomen
- Profuse vomiting with high likelihood of significant on-going fluid loss

Fluid therapy for shock should be performed using fluid boluses as described in **Chapter 3** using an isotonic replacement solution.

After normalisation of the perfusion parameters (heart rate, mucous membrane colour, capillary refill time, pulse quality, peripheral limb temperature and awareness), dehydration is corrected by intravenous fluid therapy with a balanced electrolyte solution. This should be done relatively rapidly (over 4-10 hours) if the dehydration was acute or more slowly (over 12-48 hours) if the dehydration was chronic and insidious in onset. For stable patients with self-limiting simple vomiting, oral rehydration can be done using standard oral rehydration solutions on an outpatient basis.

During hospitalisation, the on-going losses and maintenance requirements must be delivered simultaneously alongside rehydration. Ideally, on-going losses should be accurately defined, *e.g.*, by weighing the vomit. However, in most cases, this is neither possible nor realistic. Therefore on-going losses are assumed to be about half of the maintenance requirement. The maintenance requirement is calculated according to the following formula:

$$kg^{0.75} \times 70 = mL/24 h$$

For animals between 5 and 45 kg, a maintenance requirement of 2 mL/kg/h is assumed.

Electrolyte abnormalities should also be treated during or, at the latest, after rehydration. In dogs, hypokalaemia is frequently associated with vomiting. In patients with hypokalaemia, potassium is added into the fluid therapy according to the serum potassium levels (**Table 5**). It is important that the potassium-supplemented fluids are not administered too quickly. In the event that potassium-containing infusions are administered too quickly, this can cause life-threatening hyperkalaemia with bradycardia and cardiac arrest. The maximum infusion rate (0.5 mmol/kg/h) must not be exceeded. All potassium-containing infusions must also be clearly and visibly labelled.

For symptomatic treatment of vomiting, metoclopramide, maropitant and ondansetron are used. Metoclopramide works centrally as an anti-emetic and also increases gastrointestinal motility. It can be administered by the subcutaneous, oral or, for severe vomiting, intravenous routes. Intravenous delivery can be by intermittent bolus or as a continuous rate infusion. For reduced intestinal motility, e.g., for some diarrhoeal diseases, the increase in motility after administration of metoclopramide, may predispose to development of intussusception. Maropitant, a neurokinin-1 antagonist, works centrally as an anti-emetic, but has no effect on gastrointestinal motor function. Its anti-emetic effect is strong; if used in patients with undiagnosed gastrointestinal obstructions, it can reduce the symptoms of vomiting and in this way obscure the need for further evaluation and treatment. Hence, before giving maropitant, an obstructive intestinal disease should be ruled out. In animals with very severe vomiting, if vomiting persists despite maropitant administration, urgent further clarification of the cause is recommended.

Ondansetron is a powerful anti-emetic that is more effective than the above-mentioned medications. In the event that the vomiting continues despite metoclopramide and/or maropitant, ondansetron can be administered; all three medications may be used concurrently.

For protection of the gastrointestinal mucosa, proton-pump inhibitors, H_2 -receptor blockers and mucous membrane coating medications are generally used. The effectiveness and optimal combination of these medications is often disputed. At present, for dogs, famotidine and/or omeprazole or pantoprazole are used to protect the gastric mucosa by

Table 5. Potassium substitution for hypokalaemia.

Serum potassium mmol/L	K-substitution mmol/L (mL/L)	Max. rate mL/kg/h
3.5-4.0	20	25
3.1-3.5	30	16.5
2.6-3	40	12.5
2.1-2.5	60	8
< 2	80	6



Table 6. Medication and dosages of important medications for patients with vomiting.

Indication	Medication	Dose	Special characteristics
Anti-emetic	Metoclopramide	0.1-0.4 mg/kg SC every 8 h 40-80 μg/kg/h CRI*, IV	Accumulation possible
Anti-emetic	Maropitant	1 mg/kg SC every 24 h	
Anti-emetic	Ondansetron	0.1-0.2 mg/kg every 8-12 h, IV	
Anti-ulcer	Famotidine	0.5-1 mg/kg every 12-24 h, IV, PO	
Anti-ulcer	Omeprazole	1 mg/kg every 12 h, IV, PO	
Anti-ulcer	Pantoprazole	1 mg/kg every 12 h, IV, PO	
Anti-ulcer	Misoprostol	1-5 μg/kg every 6-12 h, PO	Great care should be used when handling this drug as it is an abortifacient in women
Anti-ulcer	Ranitidine	0.5-2 mg/kg every 8-12 h, IV, PO	Questionable effectiveness in dogs
Anti-ulcer	Sucralfate	30 mg/kg, PO	
Analgesic	Buprenorphine	0.01-0.02 mg/kg every 6-8 h, IV	
Analgesic	Butorphanol	0.1-0.4 mg/kg every 1-2 h, IV, IM, SC 0.1-0.4 mg/kg/h CRI, IV	
Analgesic	Fentanyl	0.002-0.01 mg/kg/h CRI, IV	
Analgesic	Metamizole	20-50 mg/kg every 8 h, IV	

^{*}CRI: continuous rate infusion

increasing gastric pH. There is no evidence that cimetidine and ranitidine lead to increases in gastric pH in dogs although ranitidine may be of benefit in some cases via its prokinetic effect. For physical protection of the gastric mucosa, sucralfate can be additionally employed. This should be administered at least two hours before or after the application of other oral therapies since it reduces absorption of many drugs (**Table 6**).

Analgesia for gastrointestinal tract pain is complex. It is usually considered to be visceral pain, and hence should be effectively managed by kappa-agonists including butorphanol. Since its analgesic effect usually lasts around one hour, butorphanol can be administered as a continuous rate infusion. In some cases, the administration of partial μ -agonists (e.g., buprenorphine) can also deliver sufficient analgesia. However, μ -agonists can lead to reduction in gastrointestinal motility. The use of a non-steroidal anti-inflammatory in patients with vomiting should be avoided due to the high potential for further damage to the intestinal mucosa. In some countries, metamizole (dipyrone) may be

used. This drug is related to the NSAIDs and has anti-spasmodic and analgesic properties. There is no evidence currently that damage to the gastrointestinal mucosa occurs with this drug if used without other NSAIDs. It is not available in some countries due to potential development of an agranulocytosis in humans.

Insufficient nutrition can lead to atrophy of the intestinal villi and, with this, to reduction of the intestinal barrier. Early nutrition can contribute to the health and convalescence of the gastrointestinal tract. For example, puppies with parvovirus that received early enteral feeding show improved albumin values compared with puppies that were not fed. Thus, in patients with vomiting, it is recommended that enteral feeding be started as soon as the patient is haemodynamically stable. The diet used should be low in allergens and highly digestible. A variety of commercial gastrointestinal diets are available, for example, Royal Canin's Convalescence support diet. In animals with severe disease, feeding may need to be supported with placement of an enteral feeding tube (see **Chapter 6**).



Ultimately, vomiting will resolve as the underlying cause resolves. Wherever possible, specific treatment to address the underlying disease should be used whether this is surgical or medical. Whilst this treatment is underway, supportive care should be used as described above. Furthermore, in some patients with gastric emptying disturbances, it is appropriate to occasionally empty the full stomach using a stomach tube. Nausea may also be improved with this and

the animal's general condition may improve. In patients where a self-limiting cause is suspected and symptomatic therapy is used without further diagnostics, regular reassessment of the patient is essential to ensure symptomatic therapy is being effective and to monitor for worsening of the disease. An initial recheck should be performed within 24 hours of discharge. In the absence of improvement, it is advised to move urgently to further diagnostics.



6. Do's and don'ts

> SUMMARY

Emergency medicine and intensive care are cross-disciplines. The clinician is faced with very diverse, serious and sometimes entirely new situations. It is essential that he/she acquires reflexes enabling him/her to react quickly while avoiding mistakes that could have dramatic consequences. The aim of this chapter is to describe what is recommended and not recommended in common situations. It covers transfusions of blood products for which group compatibility is essential, the critical animal's diet where earliness is important — with a preference for the enteral route —, as well as analgesia which plays a fundamental role in the healing process of critical animals as a result of adapted protocols, sometimes combining several molecules to reduce side effects. The rational use of extremely common medications such as antibiotics, non-steroidal anti-inflammatory drugs and corticosteroids is also described.

1/ Transfusion

Blood and its products may be used to supply red blood cells, plasma (globulins, albumin, coagulation factors) and/or platelets. Respect for proper transfusion practices is essential to ensure that transfusions are effective and safe with limited adverse reactions.

A) What to do

▶ When should a transfusion be performed?

Transfusions may be used in patients with anaemia, coagulopathies, and potentially during hypoproteinaemia. Acute or chronic anaemia (haemorrhagic, haemolytic) is the major indication for red blood cell transfusion (whole blood or packed red cells (**Figure 1**)). The decision to transfuse is based on the clinical signs shown (tachypnoea, tachycardia, heart murmurs, pallor), the haemoglobin or haematocrit (Hct < 15% (20% if acute haemorrhage) or haemoglobin < 8 g/L) and the likely chronicity.

► Transfusion compatibility must be assured

Blood types are antigens on the surface of the red blood cells (RBC) that produce an immune response when administered to a recipient.

In dogs, the DEA1 (Dog Erythrocyte Antigen) system is the most important, due to its strong antigenic property. DEA1- (negative) dogs do not have an acute reaction when transfused with DEA1+ (positive) donated blood at the time of their first transfusion, because they do not have natural antibodies against this antigen. However, if the DEA1- dog is transfused a second time with blood from a DEA1+ dog, there is likely to be a serious haemolytic reaction, due to the presence of anti DEA1 antibodies produced following the first transfusion. In cats, the A, B or AB blood types are the most important, with the majority of cats being type A. The B group is more frequent in certain exotic breeds but is found in European cats (Domestic Shorthair/ Longhair). A-type cats have low levels of anti-B allo-antibodies, which can destroy red blood cells or reduce their longevity if a B-donor is used. B-type cats have very high levels of anti-A antibodies and they can have a fatal reaction if given type A blood even after a first transfusion. AB-type cats do not have anti-A or anti-B allo-antibodies. They can receive AB, A or B blood. The MiK AG has been identified more recently. Anti-MiK antibodies may exist in certain cats, and a first transfusion may lead to a haemolytic reaction in certain patients even if a correctly typed AB transfusion is performed. Rapid and reliable blood typing tests currently exist for dogs (e.g., Alvedia Quick Test® for DEA1) and cats (e.g., Alvedia Quick Test® for A, B and AB) (Figure 2a). Cross-match tests serve to determine whether antibodies already exist in the plasma of the donor and/or recipient (Figure 2b).





Figure 1. Transfusion of whole blood in a dog suffering from immune-mediated haemolytic anemia.

Before a first transfusion, blood typing is strongly recommended in dogs and essential in cats, and a cross-match is recommended in cats. For multiple transfusions, appropriate typed blood should be used for each transfusion and cross-matches are essential for transfusions occurring greater than four days after the first transfusion.

► How to transfuse?

The transfusion must be done in optimal and aseptic conditions. The quantity of anticoagulant (typically CPDA or ACD) used is 1 mL anticoagulant per 7 mL blood. When using whole blood, a transfusion of 20 mL/kg is generally recommended and is likely to lead to an increase of approximately 10% in the haematocrit of the patient. A clinical improvement is sought rather than a target Hct value. In normovolaemic patients, the transfusion is administered over four hours. The flow rate is 1 mL/kg/h during the first half an hour, then it is increased until it is being delivered at 5 mL/kg/h. The transfusion may be administered more rapidly in hypovolaemic patients. Patients undergoing a transfusion should be rigorously monitored. Hct and

total proteins should be measured at the start and one hour after the end of the transfusion.

▶ What to do in case of adverse reaction?

Adverse reactions may be haemolytic (hypersensitivity types I and II) or non-haemolytic (volume overload, hypocalcaemia, febrile reactions, sepsis, transfusion respiratory syndrome, thrombocytopenia). In the event of agitation, discomfort, tachypnoea, vomiting or pruritus during a transfusion, the transfusion must be stopped and fluid therapy (except in the case of volume overload) as well as oxygen therapy must be provided. For type-I hypersensitivity reactions, diphenhydramine (2 mg/kg, IM) may be administered.

B) What not to do

▶ Indications and contraindications

For immune-mediated haemolytic anaemia, there is considerable risk of haemolysis of the transfused blood. However, transfusions may still be necessary to support the patient. If the benefit is considered to outweigh the risk, precautions should be increased (for example, more intensive monitoring, slower flow rate at the start). The same precautions should be taken in patients at risk of volume overload (i.e., underlying cardiac disease) and renal failure. The benefit of transfusion for the treatment of hypoalbuminaemia is controversial. Indeed, it would take 45 mL/kg of fresh plasma to increase albumin by 1 g/dL (Figure 3). Whole blood does not contain significant number of platelets. Packed Red Blood Cells (PRBCs) are ideal for the treatment of normovolaemic anaemias. Their availability is very dependent on geographical location.

Figure 2. Example of in-house blood typing in the dog using an immunochromatography technique (DEA1-) (a). Example of in-house crossmatch in the dog using an immunochromatographic technique (positive crossmatch) (b).







▶ Before the transfusion

It is strongly recommended to blood type dogs, and essential to blood type cats prior to their first transfusion. Cats must never be transfused with incompatible blood. It is better to avoid transfusing A blood and B blood to the same cat. Even if the animals have been typed, it is necessary to carry out crossmatches in case of multiple transfusions, especially if they are spaced more than four days apart.

In the absence of compatible blood, the veterinarian may be tempted to carry out a xenotransfusion of dog blood into a cat. A recent article described this practice, which may be life-saving but can be done only once and should be performed only if there is no alternative. Owners must be fully aware that this exceptional procedure has been performed.

Improperly kept blood products must not be transfused. One must avoid transfusing from incompletely filled blood bags. In that situation, the anticoagulant which contains a calcium chelator is in excess and risks provoking hypocalcaemia in the recipient.

Preventative administration of corticosteroids to avoid the formation of antibodies is ineffective. The benefit of antimicrobial therapy have not been shown.

▶ During the transfusion

Due to the risk of volume overload, all other fluid therapy is generally discontinued. Calcium-containing solutions must not be administered through the same catheter as blood products due to risk of calcium precipitation with the citrate content of the anticoagulant.

A transfusion should generally not take longer than four hours. Transfusions should only be given more slowly if the patient is at risk of volume overload and, only then, if the clinician judges this risk outweighs the risk of the blood product being at room temperature for a longer period of time. Formulas exist to calculate the volume to be transfused. However, the amount and rate of the transfusion does not depend on the calculations alone but more importantly on the clinical monitoring of the animal.

2/ Antibiotics

Continuous exposure to antibiotics (AB) and especially to broad-spectrum ones encourages mutations and resistance of bacteria in the gastrointestinal tract. Some new multi-resistant bacteria (MDR) have appeared: Methicillin-resistant *Staphylococcus aureus* (MRSA), Vancomycin-resistant *Enterococcus* (VRE), Fluoroquinolone-resistant Pseudomonas (FQRP), Vancomycin-resistant *Staphylococcus aureus* (VRSA) and Extended Spectrum Beta-Lactamase (ESBL) Enterobacteria.

Figure 3. Fresh frozen plasma.



Figure 4. Dog with septic shock in the ICU undergoing intensive monitoring and treatment including broad-spectrum antimicrobial therapy.





A) What to do

▶ Use protocols to improve the use of antibiotics

In case of sepsis or septic shock, antibiotic therapy is essential **(Figure 4).** The initial antimicrobial therapy is always empirical in nature as culture results take a minimum of two days to obtain. However, all efforts should be made to choose an antimicrobial that is most likely to be effective. Information to consider when making this choice include data from the literature, epidemiological data from local hospitals, recent patient antibiotic history, and site of the infection. In critical patients, the bacteria generally found are those of commensal flora or nosocomial bacteria. The aerobes are represented by *E. coli* (the principle gram-negative bacteria) and *Enterococcus spp.* (the principle gram-positive bacteria). However, although sometimes overlooked, anaerobic bacteria are the most numerous commensals and should not be forgotten. Once available, antimicrobial therapy should be chosen based on the results of a culture and antibiogram. The duration of administration must be as short as possible.

► Choose the antibiotics depending on their availability, their spectrum and their mode of action (Table 1)

Veterinary availability of intravenous (IV) preparations of AB varies depending on the country. ABs are classed as time-dependent or as concentration-dependent. To be effective, time-dependent ABs must be present at the site of infection while the bacteria construct their cell wall. They should be administered frequently and continuous infusions of time-dependent AB may be of benefit. On the other hand, concentration-dependent ABs have a post-antibiotic effect that prolongs the antimicrobial effects after a brief exposure to the AB and should generally be given as intermittent high doses.

► Adjust the doses depending on the patient's condition

General recommendations exist **(Table 1).** Septic shock, trauma and fluid therapy increase the volume of distribution and reduce tissue concentration of AB. As a result, the doses should be increased. Conversely, a decrease in circulating volume (dehydration) increases plasma concentration of AB. Fluid therapy is preferable to a reduction of AB doses. It is recommended that the doses be multiplied by 1.5-2 for hypoalbuminaemia. Reductions in renal clearance lower the elimination of water-soluble ABs. If clearance of a time-dependent AB is increased, it is appropriate to increase frequency of administration.

► Adequately combine the antibiotics

In situations where the use of multiple ABs is warranted, the AB mechanisms of action must be complementary and together they must provide cover against polymicrobial infections. Beta-lactams and aminoglycosides are synergistic against *Enterococcus spp.*, Enterobacteriaceae, *P. aeruginosa*, and *Staphylococci* (including MRSA). Aminoglycosides and fluoroquinolones are often combined with beta-lactams, metronidazole or clindamycin in order to target gram+ and gram- bacteria or anaerobic bacteria.

► Adopt complementary measures

It is appropriate to know the source of the infection and to do everything to control it (drainage of abscess, drainage and lavage of septic pleural effusions, laparotomy and lavage for septic peritonitis). It is likewise imperative to prevent nosocomial diseases (cleanliness of catheters, drains, urinary catheters and tracheal tubes).

Table 1. Main antibiotics IV available in veterinary medicine.

Groups	Antibiotic IV	Anaerobic effect	Spectrum	Time/ concentration	Dosage
A + Penicillin watersoluble	Amoxicillin/ clavulanic Acid	+	Gram+ Gram-	T*	20-30 mg/kg/6-8 h
Fluoroquinolone	Enrofloxacin Marbofloxacin	-	Gram+: ± Gram-: +++	T (G+) C** (G-)	Enrofloxacin Dog: 5-10 mg/kg/12 h Cat: 2.5 mg/kg/12 h Marbofloxacin 2-4 mg/kg
Sulfanomide	Trimethoprim- sulfanomide	±	Gram+ Gram-		30 mg/kg/12 h
Nitroimidazole	Metronidazole	+++	Gram+ Gram-	С	15 mg/kg/12 h over 15 min
Aminoglycoside water-soluble	Gentamicin	-	G+: ± Gram-: ++	С	5-7 mg/kg/24 h 3-5 days maximum

T*: time dependent, C**: concentration dependent.



B) What not to do

▶ Do not expose the patient to a potential AB toxicity!

Aminoglycosides are nephrotoxic and are contraindicated in cases of renal failure. If aminoglycosides are used, it is recommended that the animal also receives fluid therapy, that the drug is administered once per day preferably in the morning, and that it is not combined with other potentially nephrotoxic agents (for example, NSAIDs, ACE inhibitors, diuretics). Fluoroquinolones at doses higher than 5 mg/kg/24 h must be avoided in older cats or those with renal failure.

▶ Don't be responsible for antibiotic resistance!

Multi drug-resistance is defined as a resistance to three ABs or more to which the bacteria is usually sensitive. Improper use of AB encourages the appearance of new MDR bacteria. *E. coli* MDR would seem to be the result of the use of fluoroquinolones. Cefotaxime and Ceftazidime (3rd generation cephalosporins) could be responsible for the appearance of MRSA and MDR coliform bacteria. The use of fourth generation AB is strongly discouraged and these should be reserved for human hospital use.

▶ Prophylactic antibiotics are not recommended!

Rigorous prevention of nosocomial diseases is preferred.

▶ Do not continue ineffective antimicrobial therapy!

Discontinuing ineffective antimicrobial therapy, once culture results are received, has been associated with a reduction in hospitalisation time, costs, AB resistance and secondary infections. Short-duration treatments (three to five days) are preferred to 7-10 day treatments.

▶ Do not combine antibiotics with antagonistic mechanisms of action!

In general, bacteriostatic agents should not be combined with bactericidal agents.

► Multiple ABs should only be used concurrently if there is a strong reason to do so!

For example, evidence of a mixed microbial infection associated with a patient showing signs of sepsis or septic shock.

3/ Non-steroidal anti-inflammatories

Non-steroidal anti-inflammatories (NSAIDs) inhibit the enzyme, cyclooxygenase (COX). COX-1 ensures production of prostaglandins (PG) and as a result, the production of gastrointestinal mucous as well as supporting gastrointestinal and renal perfusion. Activity of COX-2 is enhanced during the inflammatory cascade and is the therapeutic



Figure 5. Multimodal analgesia with CRI administration using multiple syringe drivers.

target of NSAIDs. Non-selective NSAIDs (aspirin, ketoprofen, piroxicam) inhibit COX-1 and COX-2. COX-2 selective NSAIDs (carprofen, meloxicam, tolfenamic acid) have a greater effect on COX-2 than on COX-1 and "COX-2 specific" NSAIDs (deracoxib, firocoxib) have a slightly greater effect on COX-2.

A) What to do

► Use non-steroidal anti-inflammatories as antiinflammatories

NSAIDs reduce inflammatory oedema with an early effect on acute inflammation and functional recovery. For chronic inflammation, their less-pronounced effect requires prolonged administration.

Use non-steroidal anti-inflammatories as analgesics

NSAIDs act on pain originating from inflammation or lesions and on post-operative pain. In critically ill patients, opioids are generally the first choice initially. Depending on the patient's situation, NSAIDs should be considered as part of multimodal analgesia (**Figure 5**), when it is safe to do so. Numerous combinations are possible with opioids, alpha2-agonists or ketamine.

▶ Use non-steroidal anti-inflammatories as antipyretics

NSAIDs block processes triggering fever without provoking hypothermia. Certain NSAIDs are more antipyretic (tolfenamic acid) depending on their capacity to inhibit hypothalamic cyclooxygenases.

► Use non-steroidal anti-inflammatories as a platelet antiaggregants

COX-1 NSAIDs inhibit the synthesis of thromboxane and platelet aggregation. This effect is obtained with an ultralow dose and is used to reduce the risk of thromboembolism. The recommended dose



for feline thromboembolism prevention is aspirin 1 mg/kg/day, 2 or 3 times/week. The recommended dose for thromboembolism prevention in immune-mediated haemolytic anaemia in dogs is 0.5 mg/kg/day, PO.

► Protect the digestive tract!

All NSAIDs are liable to induce gastrointestinal ulcers. In dogs, the effectiveness of proton-pump inhibitors (Omeprazole, Pantoprazole) seems greater than that of anti-histaminergics ($\rm H_2$) (Ranitidine, Famotidine). It is likewise appropriate to ensure that the NSAIDs are indicated in the treatment of the disease, to use the lowest effective dose possible for the shortest duration possible, to anticipate withdrawal and accurate monitoring in the event NSAIDs are changed and to choose, in as much as possible, a selective or specific COX-2 NSAID.

B) What not to do

► Non-steroidal anti-inflammatories are contraindicated in cases of renal failure or renal hypoperfusion!

NSAIDs inhibit COX-1 and 2 reducing synthesis of PGE2, and thus encouraging vasoconstriction of the glomerular afferent arteriole as well as reduction of renal blood flow and glomerular filtration rate. The risk of acute renal failure is particularly pronounced in patients that are older, hypovolaemic (cardiomyopathy, shock, trauma) and in those treated with diuretics or ACE inhibitors. Use of NSAIDs in those groups of patients carries a real risk of acute kidney injury (AKI) which can lead to renal failure.

- ► Use NSAIDs with caution in patients predisposed to haemorrhage. Avoid salicylates!
- ▶ Do not use NSAIDs as the sole analgesic for severe to very severe pain!
- ► Avoid using NSAIDs in patients presenting with gastrointestinal ulceration or excessive production of gastric acid (renal failure)

Misoprostol (3 mcg/kg, PO every 8-12 h) lowers the risk of ulcers induced by aspirin in dogs.

Avoid combining NSAIDs with ulcerogenic medication such as corticosteroids

4/ Analgesia

Treatment of pain is fundamental in emergencies and intensive care. It must be implemented as early as possible, be multi-modal if

necessary making use of several analgesics acting on different targets, and must be tailored to the animal's condition as well as the anticipated or estimated pain level.

A) What to do

► The analgesics of choice are generally opioids

They act on the opioid receptors (mu, kappa, delta). The mu receptors are intimately involved in the modulation of nociception. Morphine, methadone and fentanyl (full mu receptor agonists) provide excellent analgesia and have the advantage of being able to be titrated to effect (**Table 2**). Opioids can be reversed (Naloxone 0.02 mg/kg, IV).

- ▶ Methadone has the same potency compared with morphine In addition, it has some NMDA antagonist effects.
- Fentanyl is about 100 times more potent than morphine
 It has a short duration of action (20 min) and is preferably administered as a continuous infusion (1-5 μg/kg/h).

► Buprenorphine, a partial mu receptor agonist, possesses a long duration of action (6-8 h) combined with moderate analgesic potential

It is difficult to titrate due to its long duration of action and its strong affinity for mu receptors. It is instead used for treatment of moderate pain.

Butorphanol, a kappa agonist and a mu antagonist, is suitable for treatment of visceral pain, especially in agitated or anxious animals

It has good sedative potential but is a weak analgesic.

- Upon admission to the Emergency Room, opioids can be combined with benzodiazepines (Diazepam, Midazolam) if a greater sedative effect is needed
- Severe pain is best managed with the implementation of a multimodal analgesic plan

Involving combinations of opioids with Lidocaine, Ketamine and/or (Dex)medetomidine delivered as a continuous rate infusion (CRI).

B) What not to do

➤ Analgesia should not be delayed upon admission to the Emergency Room, regardless of the presenting complaint (for example, dyspnoea, polytrauma, heat stroke)

Stress and agitation increase tissue oxygen consumption and the risk of hypoxia, put the medical team in danger and reduce the quality of care. Pain stimulates the sympathetic nervous system and amplifies



the response to shock. Almost invariably, the advantages of analgesia outweigh the risks.

- ► Alpha-2 agonists are to be avoided when managing care in the Emergency Room due to their depressive effects on the cardiovascular system and on renal perfusion
- ▶ NSAIDs should be avoided in the emergency context, as they may have significant effects on renal perfusion and increase the risk of gastrointestinal ulceration particularly if intestinal perfusion is compromised

They must be reserved for later phases of treatment, once the animal is stabilised.

- ▶ It is not recommended to combine butorphanol or buprenorphine with mu agonists because they act in opposition to one another
- ► Analgesia should never be withheld due to concerns about the impact on a neurological examination in patients with paresis or paralysis

Agitation and pain disturb the neurological exam more. On the other hand, benzodiazepines should be avoided if there is a suspicion of spinal trauma, because myorelaxation may be responsible for the displacement or luxation of injured vertebra.

▶ One should not fear analgesia for cases of traumatic brain injury. Analgesia reduces the elevation in intra-cranial pressure (ICP)

Opioids are commonly used. However, they may induce dose-dependent respiratory depression or hypotension. It is advised that these be titrated carefully to balance the positive and potentially negative effects. Constant rate infusion avoids analgesic peaks and troughs and reduces adverse effects. The neurological status can change quite quickly in traumatic brain injury. As a consequence, opioids with a short-duration of action (fentanyl, butorphanol) are preferred. Buprenorphine should be avoided.

▶ If using lidocaine in cats, it is important to be very aware that the toxic dose is much lower than in dogs

Table 2. Dosages of analgesic agents.

	Agents	Doses		
	Morphine	Dog: 0.1-1 mg/kg/4 h SC, IM, IV slow Cat: 0.1-0.4 mg/kg/4 h SC, IM, IV slow Infusion: 0.1-0.2 mg/kg/h		
	Methadone	0.1-0.5 mg/kg/4 h SC, IM, IV		
Opioids	Fentanyl	Loading dose: 1-5 µg/kg IV Infusion: 1-5 µg/kg/h 2-4 µg/kg/h transdermic		
	Buprenorphine	0.01-0.03 mg/kg/6 h SC, IM, IV		
	Butorphanol	0.1-0.4 mg/kg/2-4 h SC, IM, IV		
	Carprofen	Dog, cat: 4 mg/kg SID, SC, IV		
NSAID	Firocoxib	Dog: 5 mg/kg SID, PO		
	Meloxicam	Dog: 0.2 mg/kg SID, SC, IV, PO Cat: 0.05-0.3 mg/kg SID, SC, IV, PO		
Almha? aganista	Medetomidine	Infusion: 0.001-0.002 mg/kg/h		
Alpha2-agonists	Dexmedetomidine	Infusion: 0.0005-0.001 mg/kg/h		
Other	Ketamine	Loading dose: 0.5-1 mg/kg IV Infusion: 0.1-0.6 mg/kg/h		
Other	Lidocaine (dog), cautions in cats	Loading dose: 0.5 mg/kg Infusion: 1.5-3 mg/kg/h		



5/ Steroids

Prednisolone, methylprednisone and dexamethasone are the main glucocorticoids used in veterinary emergencies. Corticosteroids have anti-inflammatory and immuno-modulatory properties but show significant side effects notably hyperglycaemia, immunosuppression, Gl ulceration and digestive haemorrhage. High doses, or so-called "shock doses", do not afford any benefit in clinical improvement or survival and increase side effects. The use of corticosteroids in emergencies must always be justified and reasoned, and can never substitute good triage and major body system stabilisation. Generally, they are contraindicated for patients with diabetes, infections, gastrointestinal ulceration or in combination with NSAIDs. We have chosen to present their indications/ contraindications by emergency type.

A) Respiratory distresses

► Obstruction of the upper airways, laryngeal oedema, brachycephalic syndrome

What to do: "anti-inflammatory" doses of dexamethasone (0.1-0.2 mg/kg) or prednisolone (0.5-1 mg/kg).

► Feline asthma

What to do: "anti-inflammatory" doses of dexamethasone (0.1-0.2 mg/kg) or prednisolone (0.5-1 mg/kg). Corticosteroids may also be delivered by aerosol (for example, budesonide) and may be beneficial.

What not to do: use corticosteroids when the patient has infectious disease.

B) Cardiovascular emergencies

Corticosteroids do not have any benefit in case of cardiogenic shock or cardiac arrest. They are not recommended as part of the treatment of hypovolaemic shock.

C) Haematological emergencies

What to do: Steroids are the cornerstone of treatment for immune-mediated anaemia and thrombocytopenia (dexamethasone 0.2-0.4 mg/kg/24 h, IV) or prednisolone 1 to 4 mg/kg/24 h, IV). Transfusion may be still required to support the patient while the steroids take effect. Gastric protection (omeprazole, pantaprazole) may be required if the animal shows GI signs.

What not to do: do not use too low or too high doses of steroids. Doses should be in the immunosuppressive range. Make sure that the impact of steroid therapy on your diagnostic plan has been considered.

D) Neurological emergencies

▶ Spinal trauma

There is no objective reason to use corticosteroids.

May be done: a short anti-inflammatory treatment (prednisolone 0.5-1 mg/kg/24 h, PO) for oedema and pain reduction due to a herniated disc not associated with neurological deficiencies.

E) Traumatic brain injury

The International Brain Trauma Foundation advises against the administration of corticosteroids during cranial trauma.

F) Seizures

What not to do: Corticosteroids are neither effective nor harmless in the treatment of seizures.

May be done: Anti-inflammatory doses in animals with intra-cranial tumours associated with cerebral oedema.

G) Insulinoma

Corticosteroids (prednisolone, dexamethasone) increase blood glucose and treat hypoglycaemia.

H) Acute hypoadrenocorticism

What to do: dexamethasone (0.2 mg/kg/12 h, IV).

What not to do: administer prednisolone before the ACTH stimulation test, because these can falsely elevate the serum cortisol measurements.

I) Anaphylactic shock

Treatment of acute anaphylaxis usually depends on antihistamines, adrenaline and corticosteroids. Corticosteroids may limit any inflammatory allergic reaction.

J) Septic shock

Studies have demonstrated that a proportion of patients suffer adrenal insufficiency (Critical illness-related corticosteroid insufficiency: CIRCI) secondary to septic shock (sepsis-associated hypotension despite fluid resuscitation). It seems that low-dose corticosteroid therapy







Figure 6. Nasooesophageal tube placement (a). Tube is secured in place and an Elizabethan collar used to prevent patient interference (b).

(hydrocortisone 0.5 mg/kg/6 h, IV or prednisolone 0.5 mg/kg/24 h, IV) may be beneficial in a subset of patients.

6/ Nutrition

Nutritional support is vital in caring for animals in intensive care. However, the identification of a state of malnutrition and the implementation of a diet tailored to the animal (composition, administration route, frequency of meals) very often poses a problem in daily intensive care practice.

A) What to do

▶ It is always preferable to ask oneself if the animal has covered its metabolic needs rather than "did it eat?"

- ► The animal's feeding must be planned following the first 24 hours of hospitalisation in order to anticipate the risk of malnutrition
- ▶ One must be vigilant concerning all procedures that involve temporarily withholding food (for example, in preparation for anaesthesia, surgery, x-rays)

As these can quickly lead to a situation where the animal is being fed insufficiently.

► The current recommendations are to use Resting Energy Requirements (RER) in cats and dogs:

RER in kcal = 70 x (lean body weight in kg)^{0.75}

The nitrogen balance of an animal in intensive care is negative. It is advised to supply 30% of the RER in the form of protein (caution in case

Figure 7. Feeding via an oesophagostomy tube (a). Thoracic radiography is recommended to check the tube placement (caudal nasogastric placement) (b).









of renal disease or hepatic encephalopathy). Carbohydrate metabolism is modified in the critically ill and the carbohydrate content of the diet must be limited in order to avoid hyperglycaemia, which is a negative prognostic indicator. The main energy source should be lipids.

► Choice of enteral feeding

When the digestive tract is functional, it is strongly advised to use it. A digestive tract that does not receive nutrients experiences atrophy and dysfunction in its enterocytes. The digestive barrier becomes permeable and the risk of bacterial translocation may increase. Even if it does not completely cover the energy needs, enteral feeding should be implemented early in order to maintain the integrity and functionality of the enterocytes.

► Assisted feeding

Before feeding, it is wise to stimulate the animal through a calm environment, petting and an appetising food. Chemical stimulation may be used only short-term in order to "prime" the food ingestion. Mirtazapine (dog: 0.6 mg/kg/day, PO; cat: 3.75 mg/72 h, PO) is advised by the authors.

► Tube feeding

Naso-oesophageal tubes (**Figure 6a and b**) are positioned without "breaking" of the digestive wall; inversely, "stoma" tubes (oesophagostomy, gastrostomy, jejunostomy) require an incision of the digestive tract. Currently, the oesophagostomy is the most frequently used:

- Thoracic radiographs after placement of a naso-oesophageal or oesophagostomy tube are strongly recommended to check tube placement (Figure 7a and b).
- Re-feeding is done progressively:
 - First day: one-third of caloric need in five to six meals
 - Second day: two-thirds of caloric need in five to six meals
 - Third day: 100% of caloric need in two to four meals
- Before and after each use, apply gentle negative pressure to the tube to ensure it is empty, then instil 5-10 mL of lukewarm water into the tube to keep it from clogging.
- To promote gastric emptying and to reduce the risk of aspiration for critically ill animals that are not ambulatory the following

precautions should be taken: during and for two hours after feeding, the animal should be maintained in sternal recumbency, at all times the head should be elevated at 30°, the meal should be divided, it should be verified that the stomach is empty before each meal and a prokinetic agent should be administered (metoclopramide 0.5-1 mg/kg/day, IV).

If after 24 hours of hospitalisation, a suitable enteral feeding plan cannot be implemented, parenteral nutrition should be considered. It can be partial (peripheral venous catheter) or total (central venous catheter). The risks of septic complications, bacterial translocation due to lack of enterocytes nutrition and hyperglycaemia are considerable.

B) What not to do

► A few popular myths should be dispelled. All of the statements below are commonly used and all are incorrect:

- Nutrition is not the major problem; as long as the animal's underlying disease has been treated whether or not we give nutrition will not have an impact on its recovery.
- The animal will start eating by itself within a day or two.
- Fluid therapy nourishes the animal. If it does not want to eat, the only thing left to do is begin a parenteral feeding protocol.

► Continuing to expect a resumption of spontaneous feeding in a sick and anorexic animal is a waste of time

Avoid force-feeding (risk of stress, induction of nausea and vomiting)!

► Avoid overfeeding and giving all the RER to an animal that has been anorexic for several days!

An improperly-managed refeeding may induce serious metabolic complications such as hyperglycaemia or significant hypophosphataemia leading to haemolysis and cardiac disturbances.

▶ Do not forget to weigh the animal every day with the same scale!





Further reading

Chapter 2

Boysen SR, Lisciandro GR. The use of ultrasound for dogs and cats in the emergency room: AFAST and TFAST. *Vet Clin North Am Small Anim Pract* 2013;43(4):773-797.

Chapter 3

Mazzaferro E. Fluid Therapy for the Emergent Small Animal Patient. Crystalloids, Colloids, and Albumin Products. *Vet Clin Small Anim* 2013;43:721-734.

Davies H, et al. AAHA/AAFP Fluid Therapy Guidelines for Dogs and Cats. J Am Anim Hosp Assoc 2013;49:149-159.

Chapter 6

1/Transfusion

Bovens C, Gruffydd-Jones T. Xenotransfusion with canine blood in the feline species: review of the literature. *J Feline Med Surg* 2013;15:62-67.

Davidow B. How to give blood transfusions safely: The type and cross match. In: *Proceedings* of the 18th International Veterinary Emergency and Critical Care Society; 2012: San Antonio, USA.

Giger U. Blood typing and crossmatching in ensure compatible transfusions. In: Bonagura JD, Twedt DC. eds. Kirk's Current Veterinary Therapy XIV. St Louis, MO: Saunders Elsevier; 2009, pp. 260-265.

Tocci LJ. Transfusion medicine in small animal practice. *Vet Clin North Am Small Anim Pract* 2010;40(3):485-494.

Weinstein NM, Sink CA. Blood typing and cross matching, In: Burkitt Creedon JM, Davis H. eds. *Advanced Monitoring and Procedures for Small Animal Emergency and Critical Care*, 1st ed. Chichester UK: John Wiley & Sons; 2012, pp. 682-692.

2/ Antibiotics

Boothe DM, Silverstein DC. Antimicrobial use in the critical care patient. 2009 *Small Animal Critical Care Medicine*. Eds hopper, Silvertein. Saunders Elsevier. pp. 821-827.

Dellinger PD, Levy MM, Rhodes A, Djillali Annane D, Gerlach H, Opal SM, Sevransky JE, Charles L. Sprung CL, Douglas IS, Jaeschke R, Osborn TM, Nunnally ME, Townsend SR, Reinhart K, Kleinpell RM, Angus DC, Deutschman CS, Machado FR, Rubenfeld GD, Webb SA, Beale RJ, Vincent JL, Moreno R, and the Surviving Sepsis Campaign Guidelines Committee including the Pediatric Subgroup: Surviving Sepsis Campaign: International Guidelines for Management of Severe Sepsis and Septic Shock. *Crit Care Med* 2012;41:580-637.

Epstein SE, Mellema MS, Hopper K. Airway microbial culture and susceptibility patterns in dogs and cats with respiratory disease of varying severity. *J Vet Emerg Crit Care* 2010;20:587-594.

Hugonnard M, Chalvet-Monfray K, Goy-Thollot I, *et al.* Occurrence of bacteriuria in 18 catheterized cats with obstructive lower urinary tract disease: a pilot study. *J Feline Med Surg* 2013;15:843-848.

Plumb's, Veterinary Drug Handbook. 7^{th} Ed. Plumb DC Ed. 2012. Wiley-Blackwell. Iowa USA.

Roberts JA, Abdul-Aziz MH, Lipman JR, Mouton JW, Vinks AA, Felton TW, Hope WW, Farkas A, Neely MN, JSchentag JJ, Drusano G, Frey OR, Theuretzbacher U, Kuti JL, on behalf of The International Society of Anti-Infective Pharmacology and the Pharmacokinetics and Pharmacodynamics Study Group of the European Society of Clinical Microbiology and Infectious Diseases: Individualized antibiotic dosing for patients who are critically ill: challenges and potential solutions. *The Lancet* 2014;14:498-509.

3/ Non-steroidal anti-inflammatories

Guilford WG, Strombeck DR. Classification, Pathophysiology, and Symptomatic Treatment of Diarrheal Diseases. In: Guilford WG, Editor Strombeck's Small Animal Gastroenterology. Philadelphia: WB Saunders Co; 1996, p. 351.

Hackett TB. Gastrointestinal Complications of Critical Illness. *Vet Clin North Am* 2011;41:759-756.

Hanson SM, Bostwick DR, Twedt DC, *et al.* Clinical evaluation of cimetidine, sucralfate, and misoprostol for prevention of gastrointestinal tract bleeding in dogs undergoing spinal surgery. *Am J Vet Res* 1997;58:1320-1323.

Lanza FL, Chan FKL, Quigley EMM, et al. Guidelines for prevention of NSAID-related ulcer complications. Am J Gastroenterol 2009;104:728-738.

Luna SP, Basílio AC, Steagall PV, et al. Evaluation of adverse effects of long-term oral administration of carprofen, etodolac, flunixin meglumine, ketoprofen, and meloxicam in dogs. Am J Vet Res 2007;68(3):258-264.



Rohrer CR, Hill RC, Fischer A, *et al.* Efficacy of misoprostol in prevention of gastric hemorrhage in dogs treated with high doses of methylprednisolone sodium succinate. *Am J Vet Res* 1999;60(8):982-985.

Ross L. Acute kidney injury in dogs and cats. Vet Clin Small Anim 2011;41:1-14.

4/ Analgesia

Junot S. L'anesthésie de l'animal en urgence. *La Dépêche Technique* 2010; 122:3-9.

Sande A, West C. Traumatic brain injury: a review of pathophysiology and management. *J Vet Emerg Crit Care* 2010; 20:177-190.

5/ Steroids

Burkitt JM, Haskins SC, Nelson RW, *et al.* Relative Adrenal Insufficiency in Dogs with Sepsis. *J Vet Intern Med* 2007;21:226-231.

Burkitt JM. Reviewing corticosteroids: type, dose, and indications. *Proceedings* 13rd IVECCS 2007.

Dellinger RP, Levy MM, Carlet JM, *et al.* Surviving sepsis campaign guidelines for management of severe sepsis and septic shock. *Crit Care Med* 2004;32:858-873.

Edwards P, Arango M, Balica L, *et al.* Final results of MRC CRASH, a randomized placebo-controlled trial of intravenous corticosteroid in adults with head injury-outcomes at 6 months. *The Lancet* 2005;365:1957.

6/ Nutrition

Chan DL, Freeman LM. Nutrition in critical illness. *Vet Clin North Am Small Anim Pract* 2006:36:1225-1241.

Goy-Thollot I, Elliott DA. Alimentation du chat en soins intensifs. Encyclopédie de Nutrition Féline. Royal Canin. 2010. p.140.

Holahan M, Abood S, Hauptman J, *et al.* Intermittent and continuous enteral nutrition in critically ill dogs: a prospective randomized trial. *J Vet Intern Med* 2010;24:520-526.

Liu DT, Brown DC, Silverstein DC. Early nutritional support is associated with decreased length of hospitalization in dogs with septic peritonitis. A retrospective study of 45 cases (2000-2009). *J Vet Emerg Crit Care* 2012:22:453-459.

Mansfield CS, James FE, Steiner JM, et al. A pilot study to assess tolerability of early enteral nutrition via esophagostomy tube feeding in dogs with severe pancreatitis. J Vet Intern Med 2011;25:419-425.

Mohr AJ, Leisewitz AL, Jacobson LS, *et al.* Effect of early enteral nutrition on intestinal permeability, intestinal protein loss and outcome in dogs with severe parvoviral enteritis. *J Vet intern Med* 2003;17:791-798.

Queau Y, Larsen JA, Kass PH, Glucksman GS, et al. Factors associated with adverse outcomes during parenteral nutrition administration in dogs and cats. J Vet Intern Med 2011;25:446-452.



This book has been prepared with the greatest care, taking into account the latest research and scientific discoveries. It is recommended that you refer to the specific regulations of your country. The publisher and authors can in no way be held responsible for any failure of the suggested solutions. Evidence-based medicine has been used throughout this publication wherever possible. Where no evidence base exists, or the available evidence is conflicting or equivocal the authors have provided their collaborative opinion based on their considerable experience and expertise.

Editorial coordination: Laurent Cathalan and Alexia Kappelmann Layout: Pierre Ménard Technical Management: Buena Média Plus Pictograms: Pensiri and Nickylarson974

© 2016 Royal Canin BP 4 650, avenue de la Petite Camargue 30470 Aimargues France Tel.: +33 (0)4 66 73 03 00 – Fax: +33 (0)4 66 73 07 00 www.royalcanin.com

No part of this publication may be reproduced without the prior consent of the author, his successors or successors at law, in conformance with Intellectual Property (Article I. 112-4). Any partial or full reproduction constitutes a forgery liable to criminal prosecution. Only reproductions (Art.I.122-5) or copies strictly reserved for private use of the copier, and short quotes and analyses justified by the pedagogical, critical or informative nature of the book they are included in are authorised, subject to compliance with the provisions of articles L.122-10 to L.122-12 of the Code of Intellectual Property relative to reprographics.

